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THE
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SOME ASPECTS OF INNER EAR THERAPY.*

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St. Paul, Minn.

From the therapeutic standpoint the disorders of the inner ear have constituted for otologists a realm of defeatism and impotence. The past two decades have seen important contributions in the qualitative and quantitative analysis of inner ear disorder and have seen salient electroacoustic advances in the supplanting of accepted hearing impairment by amplifying aids. Prevention of hearing impairment has received accent, but this has been largely limited to the preservation of the middle ear in school age and the reduction of acoustic trauma in industry. The idiopathic realm of inner ear impairment while it comprises the bulk of otic disability has been largely barren of therapeutic advance. This is reasonable and right. A realm in which pathology is known but pathogenesis is unknown can hardly be graced by therapeutic advance.

Otology has long confused itself and dissipated its energy in a welter of unbasic theses of primary neuritic, heavy metallic, outdated luetic, and meaningless catarrhal hypotheses which led to the dead end of therapeutic nihilism.

*Read at the Fifty-fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., May 7, 1951.

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There is every reason to believe that an important segment of inner ear pathology translated during development into symptoms of tinnitus, hearing alteration, and vertigo results from vascular change in the end-arterial distribution of the labyrinthine artery. While some of this vascular change is predicated upon histologically demonstrable atherosclerosis, endarteritis and similar obliterative change, the usual alteration is not in vascular character but in vascular function. It represents autonomic dysfunction in the labyrinthine neurovascular sphere. The penalty for angiospastic change in the labyrinthine arterial tree is severe because the primary neurones of the inner ear are most intolerant of resulting ischemia and because the vascular anatomy of the labyrinth is ill-designed for the resorption of the transudate resulting from ischemic damage to the capillary.

SYNDROMES.

The clinical syndromes resulting from labyrinthine angiospastic change vary with the neural segment involved and the degree of involvement. They may be the picture of chronic progression due to minute, insidious and continuing neurovascular dysfunction; they may be the sudden, dramatic, all-encompassing disaster involving acoustic and static function in colossal ischemic disruption. The syndromes must be viewed and taught as variations of a single genesis — differences being due only to degree of ischemia and nature of labyrinthine elements involved. The resultant theme brings order to this vast segment of clinical otology. It points the way for therapy.

The syndromes have been elaborated before. It is well to reiterate them now before relating them to specific case histories and manner of therapy. The syndromes themselves are a matter of clinical observation. The designs of their origin are hypothetical — a graphic attempt to present a mechanism of formation for known clinical phenomena.

Primary cochlear ganglion cell ischemia (see Fig. 1) causes specific frequency tinnitus and zonal hearing impairment.

There is no distortion with diplacusis, no aural pressure, and no vertigo. It eventuates in the so-called "VIIIth nerve neuritis" or "catarrhal deafness" of yesterday.

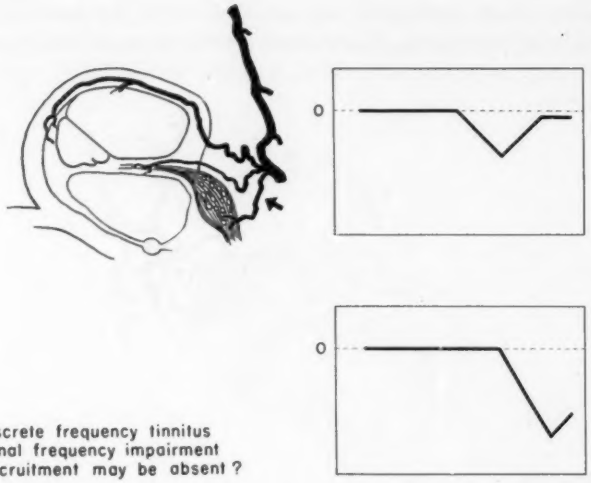
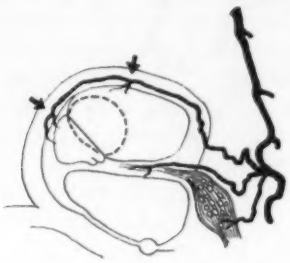


Fig. 1. Ischemia of the cochlear ganglion cells (catarrhal deafness, VIIIth nerve neuritis, etc.).

Cochlear stria vascular ischemia (see Fig. 2) results in endo- and/or perilymphatic hypertension because transudate through the damaged capillary endothelium finds itself in the labyrinthine space since the capillaries of the stria are unique in that they are covered only by the epithelium of the cochlear duct or the mesothelium of the scalae. Tonal distortion and aural pressure are experienced before hearing impairment is measurable. The presence of aural pressure and the rising curve of the audiometric tracing readily imply middle ear or tubal pathology. Thorough otologic examination and bone conduction testing are essential for differentiation. Onset vertigo is common. Tinnitus may be absent or may be of a broad frequency varying type. Without vertigo this again is the "catarrhal deafness" of old with a middle ear implication or

"the endolymphatic hydrops without vertigo" of more recent vintage. With vertigo this symptom complex ofttimes came under the coverall of "Ménière's disease" or "syndrome."

The vertigo of stria vascular ischemia results from the distortion of the vestibular neuroepithelium at the onset of lymphatic hypertension. If the latter develops exceedingly gradu-



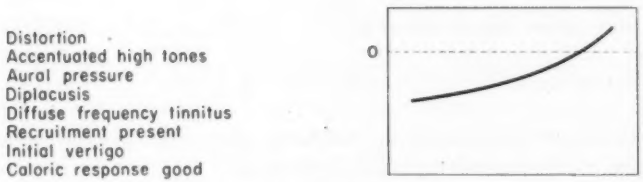


Fig. 2. Ischemia in the stria vascularis with formation of endo- and/or perilymphatic hypertension (Ménière's disease, endolymphatic hydrops **with** vertigo, labyrinthosis, etc.; or, if endolymphatic hypertension is not transmitted through utricular valve—endolymphatic hydrops **without** vertigo).

ally or is confined to the ductus cochlearis by anatomic block of the utriculosaccular valve or other device vertigo may not be experienced at onset. It is not uncommon, however, to encounter instances where lymphatic hypertension confined to the cochlear ducts for months with resultant cochlear disability suddenly diffuses through such anatomic block to the vestibular spaces with a resultant sudden vertigo. In some instances this sudden diffusion and dilution of cochlear lym-

phatic hypertension into a large space results in a decrease of tension and distortion in the cochlear duct and scalae with resultant gain in hearing following the severe vertigo (Cases IV and X).

Macular or ampullar ischemia (see Fig. 3) produces a vertigo of specific type dependent upon the neuroepithelial plaque involved. It can occur without cochlear involvement and fall

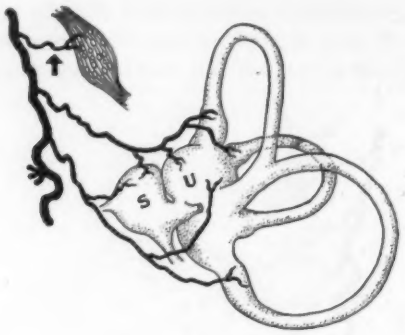
Vertigo (persistent with movement in the affected plane)
Caloric response impaired (if end-plate damage is severe and test is discriminative)

Fig. 3. Ischemia in macula or ampullary crest (pseudo-Ménière's disease).

under the former blanket of "pseudo-Ménière's disease." It is interesting and noteworthy of the punctuate character of angiospastic change in the labyrinth that, following the onset of a discrete macular or ampullary ischemia and damage the patient's neuroepithelial disability is translated for weeks or months as "dizziness" initiated by movement in the specific plane of that end-plate.

The vestibular ganglion cells distributed along the course of the vestibular nerve have a richer interlacing of arteriolar terminals than is found elsewhere in the labyrinth. Less ves-

tibular ganglion cell angiospastic ischemia may result because of this collateral support. When ischemia occurs in these primary neurones (see Fig. 4), however, the vertigo needs must be unconfined to end-plate distinction but seen in any and



Vertigo (not confined to a discrete plane of movement. Often persistent without movement)
Caloric response impaired (if neural damage is sufficient)

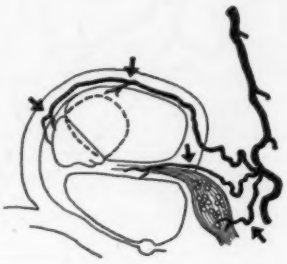
Fig. 4. Ischemia in vestibular ganglion cells (pseudo-Ménière's disease).

many planes. Until more widespread labyrinthine involvement occurs to allocate this vascular dysfunction to the end-organ there is no possibility clinically of differentiating this lesion from similar change in the central nuclear areas. It again has fallen under the looseness of "pseudo-Ménière's disease."

As in segmental angiospasm anywhere in the body, the degree and spread of neurovascular dysfunction is individual. The pure form of ischemia confined to a discrete vascular segment in the inner ear is the exception — not the rule. Clinical cases frequently tend to be predominantly cochlear ganglionic; or predominantly stria vascular; or macular or ampulla; or vestibular ganglionic, but the relative variations are unlimited. The eventual trend is for more widespread neurovascular dysfunction and the total syndromes are common (see Figs. 5 and 6).

THERAPY.

There are three therapeutic approaches which must be balanced in any case: *A.* The instigating factors of neurovascular dysfunction must be obviated. *B.* Neurovascular tone must be brought to normal balance. *C.* The agents of repair for ischemically damaged tissues must be made available.



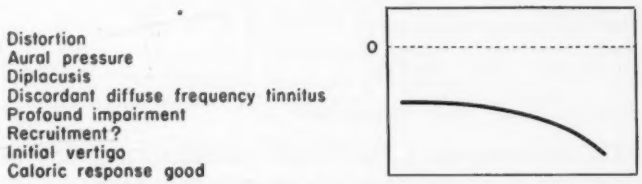


Fig. 5. Total cochlear ischemia (Ménière's disease, endolymphatics hydrops **with** vertigo, labyrinthosis, etc.; or, if endolymphatic hypertension is not transmitted through utricular valve—endolymphatic hydrops **without** vertigo).

A. The instigating factors of neurovascular dysfunction are those environmental factors to which the individual has come to react in an unfortunate, aberrant manner. Some individuals are more prone to this unfortunate reaction than others because of their inherently unstable autonomic nervous mechanisms. For this reason the family history and prior history of the victim of inner ear ischemia affords tell-tale background of other autonomic dysfunction which in the head

segments is prone to fall into the realms of vascular head and neck pain, cervical and pharyngeal myalgia, vasomotor rhinitis, or vasomotor labyrinthine ischemia.

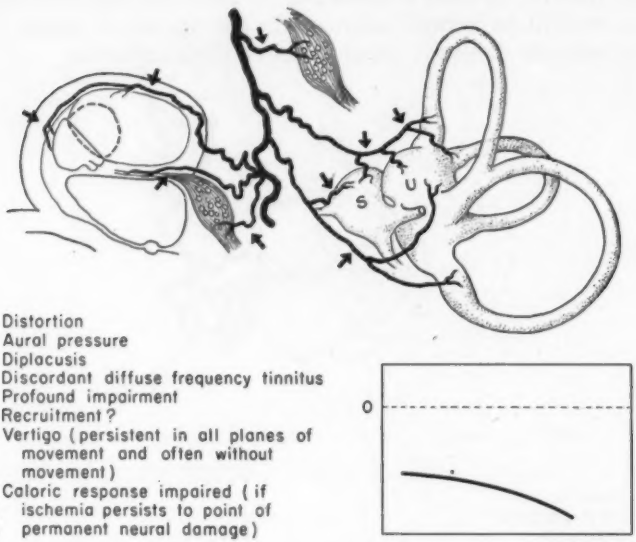


Fig. 6. Total labyrinthine ischemia (Ménière's disease, endolymphatic hydrops with vertigo, labyrinthosis, etc.).

The environmental factors which precipitate a specific neurovascular inner ear crisis are on only rare occasions autonomic-mediated antigen—antibody reactions from *inhalant or ingestant exposure* — so-called allergic crises in the inner ear. Infinitely more commonly the crisis is occasioned by unfortunate reaction to *physical environmental change.* The most common precipitor of vasomotor inner ear crisis, however, is upset in the fourth environmental sphere—*emotional reaction.*

The autonomic mechanisms are ordinarily prepared for the breakdown of crisis, moreover, by the vitiating factors of contingent stress — fatigue, coincident infection, and vasospastic agents and situations such as overindulgence in tobacco, coffee, or the pendulum-swing of excessive alcohol.

Taken individually or coincidentally these many factors impinging upon an inherently faulty autonomic mechanism conspire to the neurovascular crisis of the inner ear. *All of these factors must be weighed and neutralized as the first step in therapy.*

B. Beyond *the unmatchable tendency of nature to restore neurovascular balance* once the patient is put physiologically right with his environment there are *agents in the puny realm of drug therapy* that contribute to the same end. In general these pharmacal helps fall into two categories — *blocking agents* and *smooth muscle dilators.* The one neutralizes the neural impulse to spasm; the other actively counters spasm at the smooth muscle — principally at the arteriolar sphincter. The literature is full of haphazard and individual reference to these many agents, but cohesion and common ground is too little dwelt on.

The blocking agents — a large and rapidly growing field of interesting drugs:

1. At ganglion level there are principally tetraethylammonium chloride and direct ganglion infiltration (in our instance principally the superior cervical sympathetic ganglion with procaine, dolamine, etc.). Clinical use is limited except for the inquiring and academic mind.

2. At myoneural junction a whole host of drugs take blocking action: The so-called antihistaminics, artane, banthine, dibenamine, priscol, procaine, procaine amide, as well as atropine, hyoscine, ergotamine, etc. For the neurovascular inner ear crisis the degree of ischemia and the duration of ischemia determine the amount of permanent and residual neural damage; hence the crisis is a medical emergency. Aggressive blockade is reasonable. Our preferred agents are intravenous hyoscine as a stop-gap and intravenous procaine for prolonged infusion. To maintain the teetering neurovascular balance once a crisis is past we prefer artane and occasionally benadryl or dramamine orally.

Dosage schedules for intravenous blockade vary naturally with age, condition, and weight. Basic average doses afford some takeoff point:

1. Hyoscine hydrobromide 0.3 mg. well diluted and infused slowly intravenously is usually well tolerated.

2. Procaine hydrochloride in 0.2 per cent solution (dissolved just before use in 5 per cent glucose — or 10 per cent glucose if lymphatic hypertension is present) can be infused in total first dose of 4 mg./kg. calculated for the individual patient at a rate which is comfortably tolerated but which gives sign of useful block — slight thickening of tongue, slight feeling of float, etc. Usually this strength started at 30 drops per minute can be quickly altered in rate to accomplish this end with complete safety. The "block" should be maintained continuously for one and one-half hours to two hours once a day or twice a day as the case merits. The desired period of block naturally should be the dominant consideration, not total milligrammage in the infusions subsequent to the introductory first.

Oral dosage of artane or of benadryl or dramamine varies widely. The objective is useful block with minimum side effects. This generally entails six to eight-hour interval dose and sometimes necessitates the substitution of one drug for another because of disagreeable by-effects.

An initial 2 mg. of artane three to four times a day can be increased or decreased as need be. In instances where it is not tolerated or doesn't produce vasomotor symptom control dramamine or benadryl in recognized dose can be used alternatively.

After an angiospastic episode oral control may have to be continued for weeks or months to inhibit the usual tendency to recurrence.

It should be accented that in most cases vasomotor labyrinthine ischemia is unfortunately not seen in the acute angiospastic crisis. The otologist in his geographic orbit can edu-

catively correct this in the coming years. Unless the patient is seen during or shortly following the crisis or subsequent aggravant recurrences there is no neural residuum from the ischemia to be regained by intravenous blocking measures. To apply such aggressive therapy to established neural residua would be gross overtreatment. The patient having residua from an angiospastic crisis in the recent past deserves, on the other hand, consideration from the standpoint of a prophylactic regime designed to prevent the usual trend to future recurrence. This embraces phase one of therapy — the correction of those environmental and contingent factors that instigate neurovascular dysfunction—and continuous or intermittent oral therapy that will tend to this same end.

Smooth muscle dilating agents are more familiar in average therapy than is appreciated: nicotinic acid, roniacol, the nitrites (sodium, amyl, nitroglycerin), ethyl alcohol, trichloroethylene, papaverine, histamine, adrenalin (in physiologic concentrations), etc. Our preferred agents for the acute crisis are intravenous papaverine or intravenous sodium nitrite for the stopgap and nicotinic acid added to the procaine solution for prolonged infusion. To maintain a vasodilating tendency over an extended period of oral maintenance therapy, nicotinic acid, or, where untoward side effects prevent its use, B pyridyl carbinol (roniacol) are preferred. Modest, therapeutic doses of alcohol are useful also.

Nicotinic acid is added to the intravenous procaine infusion in a trial initial quantity of 50 mg. to the 250 cc. In subsequent infusions this amount is varied up or down in the individual to correlate "mild float" with "mild flush" as a well balanced block and dilation.

Adrenalin may be added alternatively or additionally to nicotinic acid in the infusion in amount of 0.025 mg. to the 250 cc. of diluent as a physiologic vasodilator which also has the merit of increasing heart action.

Averagely nicotinic acid or roniacol are used orally four to six times a day. To stabilize dosage in relatively unstable

reactors, they should be taken on an empty stomach, and the initial 50 mg. dose should be increased or decreased to a level giving occasional dilating sign.

C. The agents of repair for ischemically damaged tissue are both exogenous and endogenous. The basic facts of their mode of action are little understood. The first cells suffering ischemic damage are not the neurones of the end-organ but, rather, the endothelial cells lining the capillaries. The misfortune of transudate results. It can be said that early restoration of the capillary endothelium is the goal of repair therapy. The cells of the end-organ will inevitably restore within the limits of irreversible change if the integrity of the capillary is re-established.

Exogenous reparative agents are principally ascorbic acid, vitamin B factors, and vitamin E.

Asorbic acid is added routinely to the procaine infusion in amount of 1 mg. per cc. of diluent. There is evidence that it increases the tolerance to procaine and there is a mass of data available to demonstrate its effectiveness in endothelial repair.

Two hundred and fifty mg. to a gram of ascorbic acid is combined with the vitamin B complex for daily oral maintenance.

We as yet have little background for commenting on the use of vitamin E.

Endogenous reparative agents within the realm of our knowledge are principally thyroid and the steroid hormones.

Hypothyroidism of less than myzedema gravity is a clinical commonplace. It frequently is not a single entity but rather one evidence of a disordered endocrine chain. The whole picture of this complex is beyond our present horizon, but, at least, thyroid extract is a supplemental agent which is available to us, though it may not be the complete answer. When used in the rôle of a therapeutic accessory in the patient having general hypothyroid evidences it has a place. We have

found the patient's symptoms of fatiguability, chilling, sluggishness, skin thickening, lip puffiness, etc., to be more helpful in its indication than the basal metabolic rate.

We cannot yet define the place of cortisone or the adrenocorticotropic hormone. It is our impression that it will have place in aborting recurrences in stria vascular ischemia in the early stages of development of lymphatic hypertension.

During certain age periods and under some circumstances of endocrine imbalance or absence, supplemental male or female sex hormones are essential to stabilize neurovascular function. This is particularly true at the climacteric and when ear symptoms of pressure, impairment, tinnitus, etc., are exacerbated regularly in relation to the menstrual cycle.

Five to 10 mg. of methyl testosterone daily in some recurrent labyrinthine ischemic patients in the male climacteric or 0.1 to 2 mg. of diethylstilbestrol in the female climacteric can produce vasomotor stabilization not otherwise obtainable. Where the vasomotor aural symptoms aggravate consistently in the last two weeks of the menstrual cycle, 10 mg. of methyl testosterone daily during this period for one or two consecutive cycles can be corrective.

Specific endocrine dosage varies so widely with the patient and the product that the individual otologist should provide himself with a background of his own reading or from his consultant endocrinologist before embarking on this phase of treatment.

CASE REPORTS.

Following are reports on cases illustrating the various types and combinations of types of vascular ischemia of the inner ear. Special otologic examination made repeatedly in each case during the course of the disorder ruled out the middle ear as the source of symptoms and impairment by means of frequent tubal recheck and tuning fork and/or bone conduction audiometric testing.

Where pertinent systemic physical conditions coexisted with the labyrinthine disability notation will be made. General examination including cardiovascular system, serology, fundi, cranial nerves, etc., may be assumed to present no related findings unless specifically mentioned. Pertinent reference to situations of physical or emotional stress seemingly so important in the production of or exacerbation of these conditions will be occasionally made.

Since in most of these cases during the acute phase of the vasomotor crisis repeated intravenous injections of 250 to 500 cc. of a 0.2 per cent solution of procaine hydrochloride in 5 or 10 per cent dextrose solution with one-twentieth mg. of adrenalin hydrochloride and 500 mg. of ascorbic acid were used, reference to such therapy for purposes of brevity in the case reports will be merely as "intravenous procaine." In some of the cases 50 mg. of nicamin replaced the adrenalin in the mixture, and where such was the case due notation will be made. Many other oral and intravenous vasodilators and blocking agents have also been used as substitutes for or in conjunction with intravenous procaine. We feel that each of the pharmacologic agents mentioned on the previous pages have due place in inner ear therapy. One cannot be used routinely to the exclusion of the others.

Vasomotor Ischemia of the Cochlear Ganglion Cells (see Fig. 1).

Case 1: A. G., female, age 23, complained of steady tinnitus in the left ear of several weeks' duration. Her middle ears were normal. The tinnitus was similar in tone to the high frequencies which were very minimally down on the audiogram (see Fig. 7, upper). She was given one intravenous procaine injection. The tinnitus disappeared and did not return. Fig. 7 (lower) shows the audiogram four days later.

Comment: The amount of involvement here is minimal and such cases are frequently neglected because of the lack of associated hearing loss or vertigo. As ischemia increases in severity and extent, permanent disability develops.

Case 2: This is a more severe example of cochlear ganglion cell ischemia.

A. R., male, age 45, had suffered from chronic vasomotor rhinitis for 20 years. On March 30, 1950, the patient complained of tinnitus of one day's duration in the left ear. The audiogram (see Fig. 8, upper) showed significant inner ear impairment on the left. He was given three intravenous procaine injections plus oral niacin during a period of 10 days. By April 10, 1950, the tinnitus was gone, but the audiogram (see Fig. 8,

lower) was still somewhat down. Oral niacin was continued. He drove much of the subsequent night through a storm and the following night attended a party until midnight. The next morning after this fatiguing activity he had sharp tinnitus on awakening, and his audiogram was down again (see Fig. 9, upper). Intravenous procaine promptly relieved the tinnitus except for minimal background noise. After two additional intravenous procaine injections he had to listen very carefully for the finest of tinnitus and his hearing was virtually normal (see Fig. 9, lower). He was advised to continue oral niacin for a safety marginal six weeks and to use it intermittently thereafter at premonitory sign of vasospastic recurrence.

Vasomotor Ischemia in the Stria Vascularis (see Fig. 2).

Case 3: A long standing repeatedly troublesome case for which labyrinthotomy or streptomycin destruction of the labyrinth was proposed. Fortunately, however, changing to a less rigorous and tension-producing job has alleviated her symptoms.

T. R., female, age 42, was first seen on April 8, 1948. She gave the history of onset of bilateral tinnitus and pressure sensation in the ears without vertigo in July, 1947. At that time she was seen at another office and this audiogram obtained (see Fig. 10, upper). She was treated by inflation and nicotinic acid during the winter months of that year with improvement in the tinnitus on the right but persistence on the left. She was first seen by us seven months later (see Fig. 10, lower). Examination revealed her drum heads and middle ears to be normal. After eight intravenous histamine injections plus oral niacin the tinnitus was less, although it was still quite constant on the left, and the hearing was improved (see Fig. 11, upper). Subsequent to an extreme chilling, she developed a sharp increase in the tinnitus (see Fig. 11, lower). Communication with her internist revealed he had previously given her thyroid, because of other hypothyroid symptoms. She was told to resume thyroid as well as to continue her oral niacin, and drop an overload of extracurricular activities. She was next seen seven months later, at which time she had only occasional tinnitus in the left ear when exposed to cold. An amazing return of acuity was noted (see Fig. 12, upper).

Six months later on return examination she complained that she had for some time noted fluctuating but continuous tinnitus, diplacusis, aural pressure, and distortion of sound in her left ear. Symptoms usually increased just before and during her menstrual period. Mild vertigo and some peculiar nausea and vomiting were recently present. She had had some depreciation of hearing in the right ear as well as the left (see Fig. 12, lower). She was given daily intravenous procaine and one-tenth mg. of diethylstilbestrol daily orally, together with ascorbic acid, vitamin B complex, and niacin. In less than a week her head symptoms had cleared completely, but several days later in bearing an overload of special responsibilities she developed a roar in her left ear, distortion, and pressure sensation again. She again was given daily intravenous procaine injections in addition to her oral medications and 1 cc. mercuhydrin was added intramuscularly every fourth day in an effort to reduce the lymphatic hypertension more promptly. After nine intravenous injections her ears had cleared (see Fig. 13, upper). A month later her hearing dropped down again to the 30 db level in spite of the continued use of her oral medications. After five intravenous procaines the hearing was normal and diplacusis gone.

After a vacation trip to Europe she returned to her strenuous work and in a matter of weeks she again developed tinnitus, this time bilateral,

with aural pressure and hearing impairment in the left ear (see Fig. 13, lower). There was no vertigo. She also at this time developed asthma for the first time in her life. Oral niacin, ascorbic acid, diethylstilbestrol, and B complex hadn't prevented the return of ear symptoms. She was told to discontinue the vasoconstrictor histaclopane which she had been using for her asthma and was given 100 mg. of cortisone daily for four days. Four days later her asthma was vastly better. She was maintained on cortisone daily for three days and then on alternate days for an additional three weeks, but the lymphatic hypertension with tinnitus, hearing impairment, and aural pressure was unrelieved.

One month later she was hospitalized for two days because of an acute vasomotor upset — this time in the right labyrinth — with pressure sensation, tinnitus, and vertigo. She was readily controlled by procaine, but symptoms recurred in a week. She was again controlled with two intravenous procaine injections. She continued oral maintenance thereafter with artane, niacin, and diethylstilbestrol.

Because of these later two episodes of vertigo in the face of her continued use of oral medications she sought consultation elsewhere and streptomycin destruction of the labyrinth or labyrinthotomy was suggested. This was not followed through, however. Instead she continued oral medications, and prophylactically took, in addition, sodium nitrite 100 mg. intravenously at the suggestion of trouble. At long last she acceded to our repeated insistence that she get rid of her duties as a charge nurse. Six weeks after surgery had been suggested and refused it was apparent that her lighter program and oral medications were relieving the lymphatic tension (see Fig. 14, upper). She felt in excellent general health, and the tinnitus and aural pressure were lessening. She is maintaining on 3 gr. of thyroid daily, niacin, B complex and diethylstilbestrol. When on rare occasions she feels the slight nausea which usually precedes her vertigo she checks it with artane. Her audiogram is practically at normal now (see Fig. 14, lower).

Case 4: This case of vasomotor ischemia in the stria vascularis will be briefly discussed without detailed reference to day to day therapy, as it was in general quite similar to that used in the previous case. Note again the episodes of marked depression of hearing in the lower tones with return to normal even after prolonged periods of time.

E. T., female, age 60, was first seen in June of 1948, complaining of tinnitus, diplacusis, and hearing loss in the left ear (see Fig. 15, upper). This had been present for five days and had been associated with vertigo, nausea, and vomiting at the onset. She gave a history of a similar episode occurring two years previously which had cleared spontaneously over a period of many months. This time she was given intravenous histamine treatments and oral nicotinic acid and cleared of her symptoms during the next two weeks. She remained clear for another two years (see Fig. 15, lower) save for an occasional transient minor episode of vertigo sometimes associated with minor temporary tinnitus.

In May of 1950 she had an abrupt return of tinnitus and aural pressure in the left ear (see Fig. 16, upper). Daily intravenous histamine and procaine injections were used alternately. About two weeks after the onset of the tinnitus and auditory block the patient experienced a severe spell of vertigo lasting for several hours, after which her ear seemed much cleared and the tinnitus ceased (release of lymphatic hypertension through the utriculosaccular valve?). The hearing was dramatically improved (see Fig. 16, lower). Unfortunately a few days later the roar-

Fig. 7—Upper.

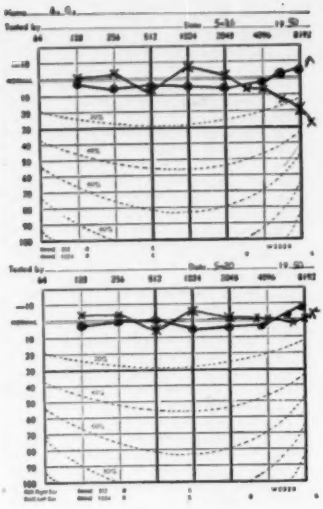


Fig. 7—Lower.

Fig. 8—Upper.

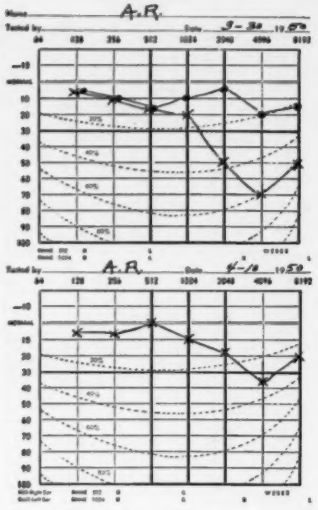


Fig. 8—Lower.

Fig. 9—Upper.

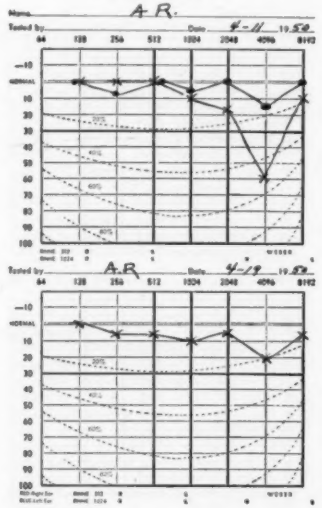


Fig. 9—Lower.

Fig. 10—Upper.

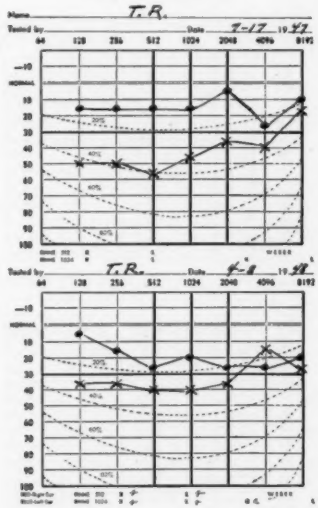
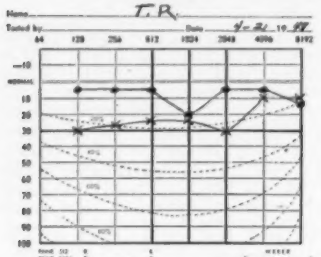


Fig. 10—Lower.

Fig. 11—Upper.



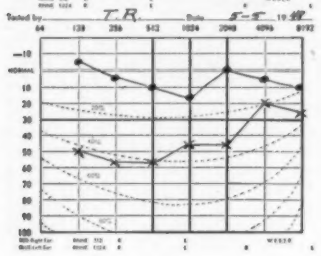
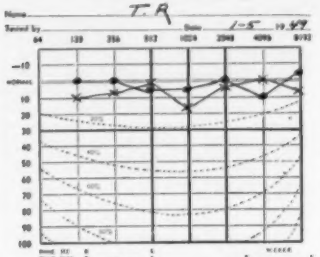


Fig. 11—Lower.

Fig. 12—Upper.



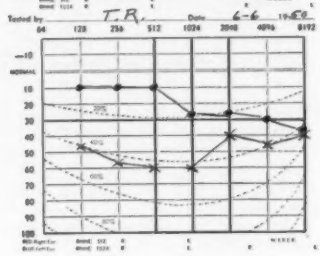
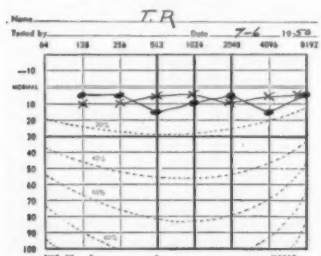


Fig. 12—Lower.

Fig. 13—Upper.



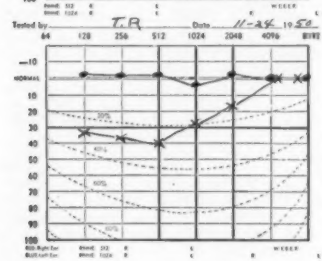
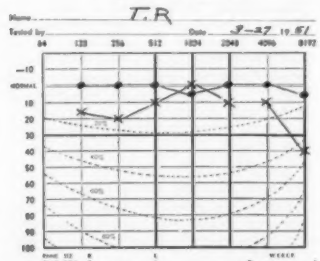


Fig. 13—Lower.

Fig. 14—Upper.



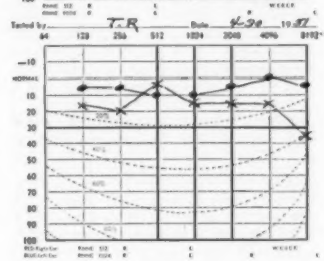
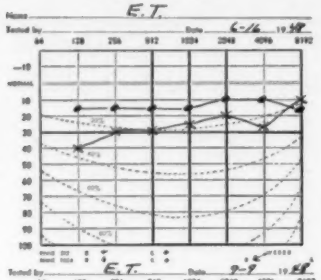


Fig. 14—Lower.

Fig. 15—Upper.



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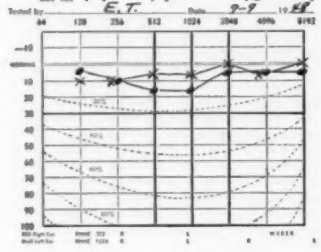

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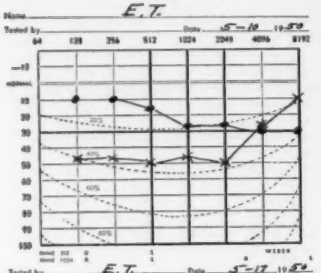

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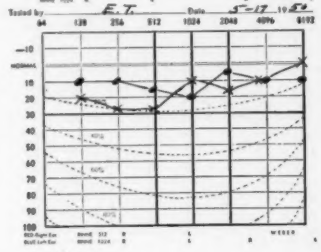
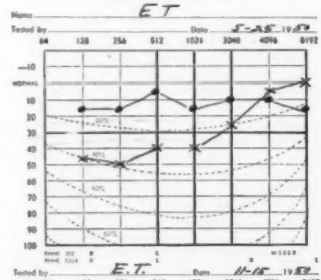


Fig. 17—Upper.



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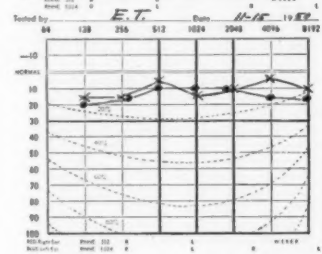
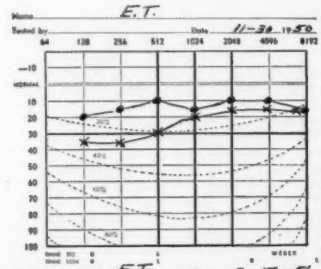


Fig. 18—Upper.



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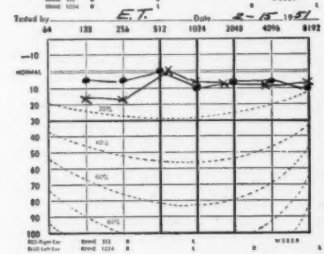

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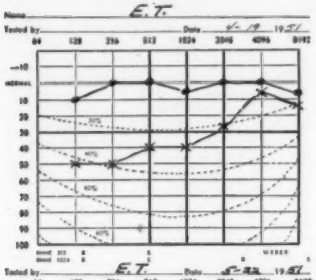


Fig. 19—Lower.

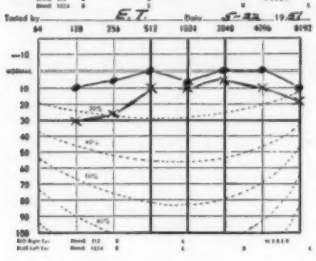
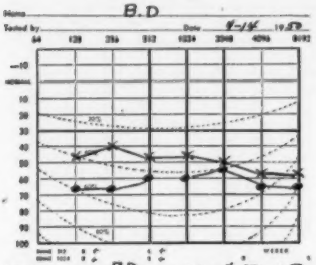


Fig. 20—Upper.



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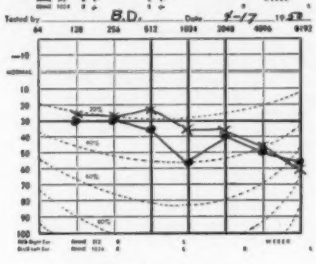


Fig. 21—Upper.

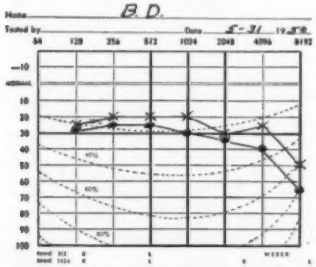
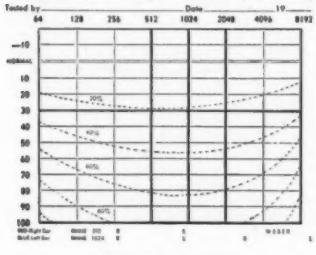


Fig. 21—Lower.



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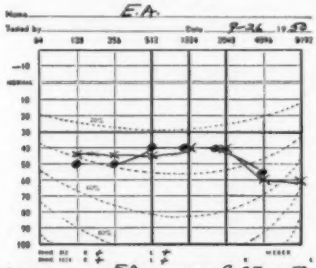

Fig. 22—Lower.

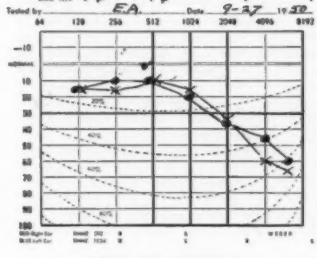

Fig. 23—Upper.

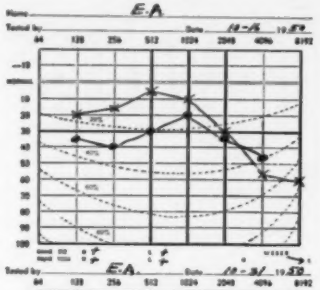

Fig. 23—Lower.

Fig. 24—Upper.

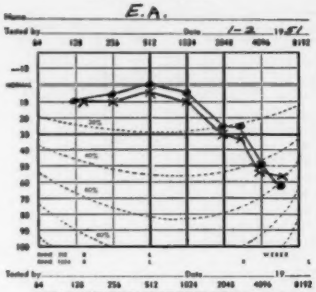

Fig. 24—Lower.

Fig. 25—Upper.

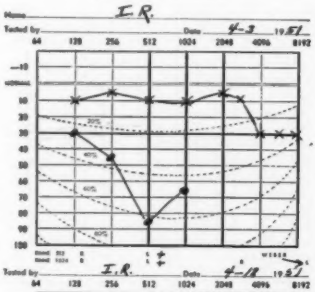

Fig. 25—Lower.

Fig. 26—Upper.

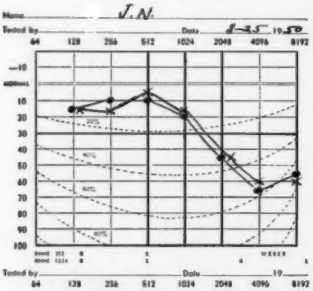

Fig. 26—Lower.

Fig. 27—Upper.

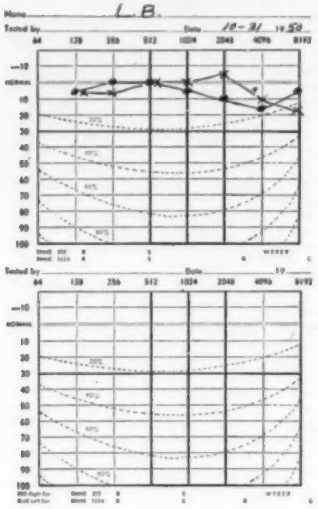

Fig. 27—Lower.

Fig. 28—Upper.

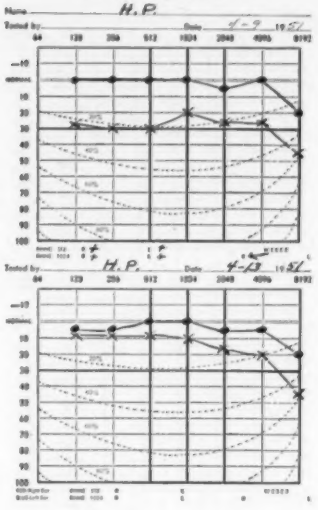

Fig. 28—Lower.

Fig. 29—Upper.

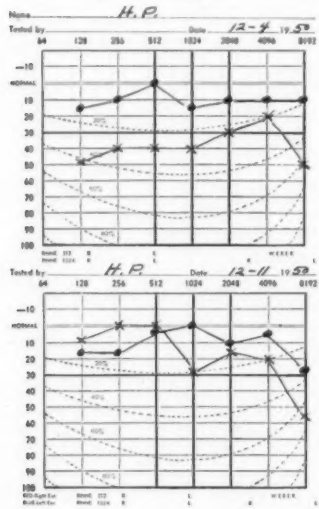

Fig. 29—Lower.

Fig. 30—Upper.

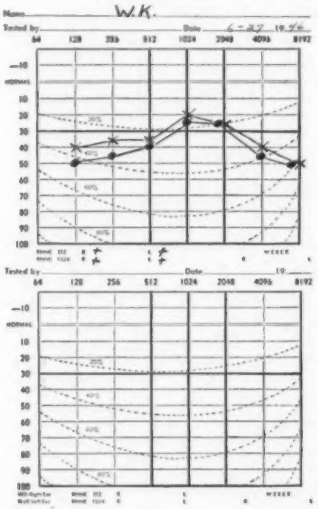

Fig. 30—Lower.

Fig. 31—Upper.

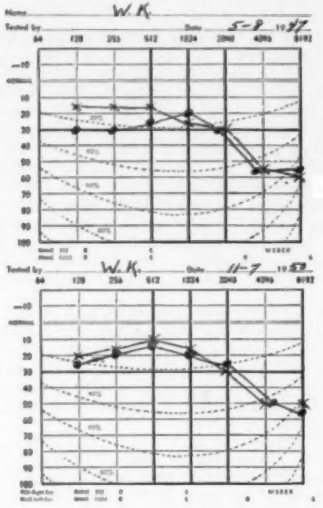

Fig. 31—Lower.

Fig. 32—Upper.

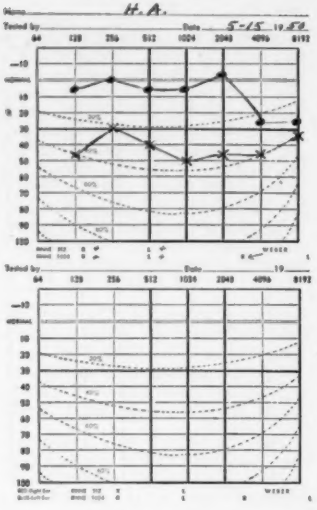

Fig. 32—Lower.

Fig. 33—Upper.

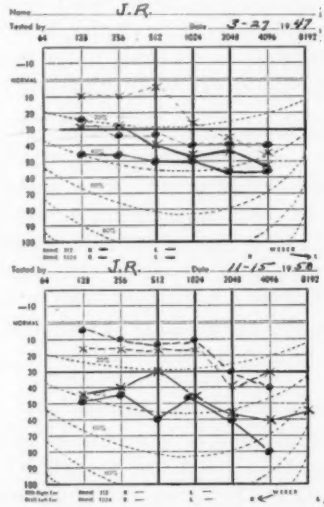

Fig. 33—Lower.

Fig. 34—Upper.

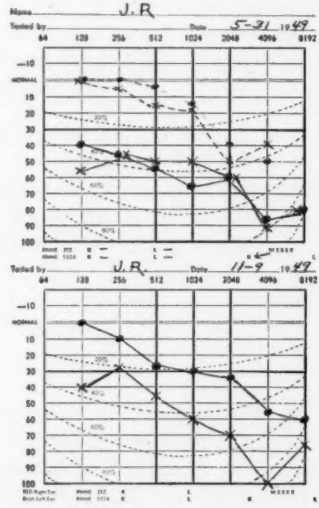

Fig. 34—Lower.

Fig. 35—Upper.

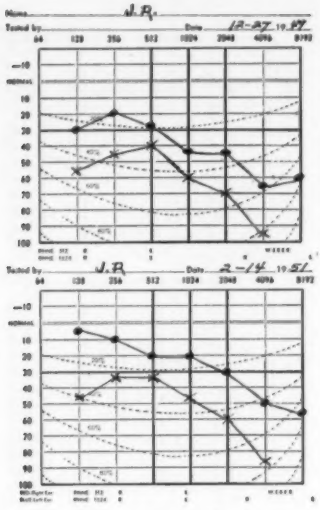

Fig. 35—Lower.

ing returned (see Fig. 17, upper). She improved during the Summer, but during the Fall ear symptoms developed again, only to clear by November (see Fig. 17, lower) after a severe prolonged period of mucous colitis with severe diarrhea. Whether the dehydration resulting from the dysentery or ACTH and cortisone, which were used at this time, were responsible for the excellent return of hearing was impossible to say. Two weeks later tinnitus and vertigo recurred, however (see Fig. 18, upper). By February the audiogram was again normal (see Fig. 18, lower) with a complete absence of tinnitus. Six weeks later the tinnitus, vertigo, and blocking again recurred. During the next few weeks the ear cleared somewhat, only to recur again with initial vertigo, aural pressure, and hearing impairment (see Fig. 19, upper). It has since gradually improved with treatment so that now only faint tinnitus is present (see Fig. 19, lower).

In virtually every exacerbation in this long and recurring course a period of emotional trial preceded recurrence — often accompanied also by an episode of mucous colitis. Her vertigo is usually only initial with each recurrence, as is usually the case in endo- or perilymphatic hypertionsion. Consequently, and in view of the potentially normal cochlea present, therapeutic labyrinthine destruction would be a grievous error.

The patient is being maintained on oral niacin 100 mg. four times a day one-half hour before meals and at bedtime (a mildly flushing dose for her), potassium chloride (Enseals) one-third gm. three times a day, diethylstilbestrol 0.1 mg. daily, and thyroid 60 mg. daily, plus vitamins B and C.

Comment: These cases are not exceptional in average otologic experience. They demonstrate the remarkable salvage in hearing which is possible when the lymphatic tension in the labyrinth is relieved. It may recur and last for prolonged periods of time, but a good prognosis for recovery is present if the high tones are essentially uninvolved.

Because the flat and rising audiometrical curves of lymphatic hypertension, together with the symptom of aural pressure superficially implicate the middle ear, these cases require extreme differential care. Accordingly we have in each instance had confirmatory examination and testing done independently by three competent otologists. In fact, Case 3 has been examined over a period of four years by six different otologists to confirm the inner ear source.

The next two cases represent a picture of lymphatic hypertension but have an uncorrectible high tone loss superimposed. Whether or not the high tone ganglionic loss in these cases resulted from ganglion cell ischemia occurring simultaneously

with the lymphatic tension of stria vascular ischemia or independently as previous ganglion cell ischemia (as depicted in Cases 1 and 2) is impossible to say.

Case 5: B. D., female, age 69, when first seen on April 14, 1950, complained that for the past six months she had had a hearing problem which started in the right ear but later involved the left. It began with roaring and buzzing in the right ear, severe vertigo, nausea, and vomiting. Elsewhere she had been given a salt-free diet and nicotinic acid. Three months before examination the left ear had become similarly involved. She frequently experienced vertigo of mild degree associated with an increase in the ever present tinnitus and a disturbing feeling of fullness in the ears. In addition to hearing impairment, aural pressure and tonal distortion were extremely distressing. The audiogram showed marked loss of hearing (see Fig. 20, upper). The caloric response was normal on both sides and reduplicated the sensation she had had during her milder attacks of vertigo.

Intravenous procaine completely relieved the tinnitus on the first day it was used. After intravenous procaine on three successive days tonal distortion and aural pressure were relieved (see Fig. 20, lower). In the succeeding six weeks she had nine additional intravenous procaine injections and daily oral niacin, ascorbic acid, and B complex. The hearing improved (see Fig. 21) and there was no further pressure or vertigo.

She was advised of the possibility of exacerbation under the strain of her teaching program, and warned to lighten her load at first signs of recurrence and resume oral niacin and artane for a prophylactic three weeks under such circumstance.

Case 6: E. A., female, age 68, never had much difficulty with hearing until two days before examination. Then, during an anxious, nervous period she had a block develop in her ears with a feeling of pressure and marked distortion of sound plus a steady pulsing noise bilaterally (see Fig. 22, upper). She had also had a persistent sensation of vertigo of moderate degree for the two days. Examination revealed both middle ears to be clear. Blood pressure was 190/100. Her cranial nerves other than the VIIIth were intact. She was given 1 cc. of papaverine intramuscularly and oral niacin. The next day her hearing was markedly improved (see Fig. 22, lower). She has been continued on oral papaverine and niacin. She has occasional episodes alternating from one ear to the other (see Fig. 23, upper and lower) of tinnitus, aural pressure and mild vertigo which have been aborted with intravenous papaverine; however, her general course has been satisfactory, as shown in her most recent audiogram (see Fig. 24). This in spite of blood pressure elevation at times up to 260/140.

Case 7: This is a case of *total cochlear vasomotor ischemia* (see Fig. 5). As the case study will show, the prognosis for the return of function in such cases is poor. This is especially true if the hearing for the higher frequencies is *nil* when first seen following the vasomotor crisis.

I. R., male, age 53, was first seen on April 3, 1951, complaining of hearing loss and noise in his right ear (see Fig. 25, upper). Eleven days previously the patient had been at cemetery services where a wind blew against his unprotected right ear for a prolonged time. Thereafter he noticed his hearing drop within a matter of hours, and he developed a broad confused noise in the right ear. He had noticed no severe vertigo.

but had had a minor episode which made him careful of rapid movements. The sensation of fullness and pressure in his ear was distressing. General health had been good previously. He had been seen elsewhere on the day after onset and had been started on nicotinic acid and roniacol tartrate without improvement.

Examination revealed normal drum heads and middle ears. There was loud voice perception only inconstantly at contact in the right ear when the left was masked. The caloric responses were equal bilaterally. He was given intravenous procaine with 50 mg. of nicotinic acid per 250 cc. of diluent and was continued on oral niacin, roniacol tartrate and was given artane and vitamins B and C in addition.

By April 9, 1951 — after five intravenous procaine injections — he heard soft voice at farther than arm's length in the right ear. There was less pressure and tinnitus.

On April 12, 1951 — after nine intravenous procaine with nicamin injections — he heard voice (although not recognizable) over the telephone for the first time.

There was no further improvement with subsequent vasodilating therapy (see Fig. 25, lower). The patient is being maintained on oral nicotinic acid, vitamin B complex, and ascorbic acid because his nervousness and depression over the episode frequently excite a transient aural pressure and fine tinnitus in the opposite ear. He has many symptoms suggestive of the male climacteric and maintains much better general tone with 5 mg. of methyl testosterone sublingually twice a day.

Comment: This case points up the medically emergent character of labyrinthine ischemia. Ganglion cells deteriorate quickly in the presence of severe ischemia. Hours are important. Delay of days and weeks in correction of ischemia usually results in severe, irreversible disability.

The vestibular portion of the labyrinth as a whole or its individual segments can be involved by vasomotor ischemia.

Ischemic impairment of one ampulla is not uncommon (see Fig. 3).

Case 8: J. N., male, age 63, had had an abrupt attack of vertigo with nausea and vomiting two months before examination. He had experienced no fullness of the ear, tinnitus, or hearing loss. He had had no further spontaneous attacks since the initial one, but ever since had a most distressing sensation of vertigo when he moved his head in the anterior vertical plane, as when looking upward.

In the face of this persistence one must presume residual ischemic damage to the neuroepithelium of the crista of the anterior superior semicircular canal. It was of a degree that was symptomatic, yet not demonstrable by the crude inadequacy of the caloric test. His audiogram (see Fig. 26) showed a bilateral high tone hearing loss which was pre-

sumably old. He was placed on vasodilating therapy orally as a precaution against ischemic recurrence. So far as the defective signal from the damaged end-plate was concerned the patient was reassured that in due course the balances of compensation and suppression would rid him of the positional annoyance.

The utricular vascular supply may be involved singly (see Fig. 3). A strong sensation of pulsion results. If ischemia is severe enough to damage the macular end-plate such an affected individual maintains, during the compensatory period, a feeling of pitching forward while in motion as though being pushed.

Case 9: L. B., female, age 47, had the sensation of falling forward whenever she was in motion for several weeks following a sudden severe vestibular attack associated with nausea and vomiting with no apparent hearing loss (see Fig. 27) or tinnitus.

Comment: The problem in an instance of this sort is whether to allocate the lesion to the end-organ or to the central nuclear area, since there is no localizing cochlear symptom. The problem seems resolved, however, when one appreciates the extraordinarily unlikely fact of discrete plane involvement in the diffuse anatomy of the nucleus.

Where more than a single crista or macular plaque is involved in the vestibule the residual vertigo is more diffuse and not limited to production in only one place of motion. This, of course, is the more common picture.

Case 10: The possibility that the *utriculosaccular* valve described by Bast and Anson and others can suddenly release endolymph under pressure from the cochlear to the vestibular side of the labyrinth is illustrated by H. P., female, age 42, with a past history of fluctuating hearing in the left ear, who developed a marked hearing loss with no perception of loud voice which persisted for three days and which suddenly "cleared" markedly in a matter of hours coincident with a sudden attack of vertigo. Examination 24 hours after the vertigo showed what she regarded now as a comparatively "clear" left ear to be still significantly impaired (see Fig. 28, upper). Apparently the unmeasured impairment which had "cleared" to this level must have been originally very severe. Within a week on oral nicotinic acid, thyroid and diethylstilbestrol therapy there was excellent return (see Fig. 28, lower). This same patient, nine months before, had had an equally severe lymphatic tensive impairment (see Fig. 29, upper) persist for five months without treatment and then clear to normal hearing promptly after taking nicotinic acid, thyroid and diethylstilbestrol orally for a week at that time (see Fig. 29, lower).

Case 11: That prolonged vasodilator therapy can prove useful in *preventing gradual progressive hearing loss* and in regaining the recent increment of impairment in a vasomotor cochleitis is evidenced by W. K., male, age 60, who was first seen on June 27, 1946, complaining of progressive loss of hearing and bilateral tinnitus. He had had chronic vasomotor rhinitis and bronchitis for over 20 years, exhibiting marked sensitivity to interior dust and to physical change. During the four months

preceding examination he and his intimates had noted a disturbing increase in the rate of decline of his hearing (see Fig. 30). He has been taking oral nicotinic and vitamin B complex since that time. His tinnitus is now only occasional and his hearing has improved during the last five years (see Fig. 31, upper and lower).

Case 12: Vasospasm is not limited to the labyrinthine arterial bed. This is illustrated in a case with *simultaneous cerebral and labyrinthine vasospasm.* H. A., male, age 47, was first seen on May 15, 1950, with a complaint of recurrent attacks of vertigo and unconsciousness. These dated back two years. The first attack had left him hard of hearing in the left ear with constant tinnitus on that side. He was unconscious for two or three hours at that time. The attacks since had occurred as often as several times a month with severe vertigo the prominent symptom. Unconsciousness frequently but not always accompanied the vertigenous attacks and was associated with numbness of the right hand and leg. Neurological examination had been negative. His audiogram showed a flat 50 db loss in the left ear (see Fig. 32). Caloric testing showed a minimal residual response on the left with massive douching. The reaction on the right was somewhat delayed but in the main normal.

History revealed that previous to the onset of his illness two years ago he had done well running a department store in South Dakota. At that time he sold out and bought another store in Minnesota. He then was caught in an effort at expansion in the building jam of the postwar period. It was at this time that the ear and cerebral symptoms developed.

The patient's internist had first treated him aggressively with intravenous histamine and oral vasodilators and had then had neurological and neurosurgical consultation relative to an VIIIth nerve section. The neurosurgeon had suggested labyrinthotomy as a more satisfactory procedure.

It was felt that labyrinthotomy would be purposeful to control the vertigo and that there was little harm in destroying the slight cochlear reserve, but that it would hardly control the intracranial vasospasm. Accordingly he was urged to retire or unload his business pressures in some manner in an effort to control both his labyrinthine and his intracranial vasospasms, rather than have the labyrinthotomy done to answer only half his problem. He was given an oral maintenance dose of nicotinic acid, artane, and phenobarbital.

Since he complied with this advice he has had only two very minor momentary attacks of vertigo and no unconsciousness. His hearing level remains reduced to the same degree and the caloric response on the left remains virtually absent.

Vasomotor changes occur in the inner ear of the otosclerotic, too, and an understanding of this fact may aid in the selection of suitable fenestration candidates, and may explain some of the eventual failures of fenestration surgery.

Case 13: J. R., female, age 42, is an *otosclerotic* who was judged unsuitable for *fenestration surgery* in 1946 because she had had recurrent attacks of vertigo and exacerbations of right ear tinnitus for two years. Her first examination audiogram (see Fig. 33, upper) showed the picture of otosclerosis with superimposed labyrinthine stria vascular ischemia (note the depression of the bone conduction on the right side). Pyribenzamine,

a capsule combining amytal, scopolamine, and atropine, and intravenous histamine were used at various times during 1946 to control the vertigo. In May of 1947 severe headaches of several days' duration associated with increased roaring in the ears were immediately controlled by 3 cc. of tetraethylammonium chloride intravenously on two occasions. Extensive effort was made to have her curb her ambitions and perfectionisms and live within her emotional means. During 1948 she was comfortable save for mild seasonal pollenosis. She was also comfortable during 1949 and 1950 and an audiogram at that time (see Fig. 33, lower) revealed absence of the previously lowered bone conduction on the right.

Comment: In view of the absence of lymphatic hypertensive symptoms during the last three years, this patient might now be considered more seriously for fenestration surgery, although she does have a basic systemic vasomotor instability (headache, pollenosis, labyrinthine ischemia) which might at any time jeopardize the benefit resulting from the surgery.

Case 14: The postfenestration effects of a vasomotor reacting inner ear are shown by J. R., female, age 40. She was first seen on May 31, 1949, complaining of hearing loss in both ears of 12 years' duration. Tinnitus was present bilaterally. No vertigo was complained of. An audiogram (see Fig. 34, upper) showed markedly reduced hearing bilaterally. Bone conduction audiometry revealed the 2,048 frequency and those higher to be markedly depressed. Tuning fork tests confirmed the bone conduction audiometry. She was a borderline surgical case. A fenestration was performed on the right ear on June 22, 1949. Postoperatively her course was good and on Nov. 9, 1949, her hearing recorded an average improvement of 25 db in the operated ear (see Fig. 34, lower).

On Dec. 27, 1949, she complained of a sensation of pressure, sound distortion, and hearing impairment which had been present in her operated ear for 24 hours. An audiogram (see Fig. 35, upper) revealed the hearing in the operated (right) ear to have dropped off about 10 db from its previous level. The fenestrum was patent. The patient had had a nervous, anxious 48 hours preceding the onset. This period of anxiety had terminated and it was decided to give her natural corrective neurovascular processes opportunity to clear the ear.

One month later an audiogram revealed the original postoperative level regained; however, six weeks after this she was given oral nicotinic acid and B complex to be used during symptomatic periods because she had come to note that at the time of her menses or frequently at times when she was tired or nervously upset she would develop tinnitus and aural pressure in her operated ear. In an enthusiasm of overdoing, she used the medications most of the time. It controlled her symptoms well. Her aggravant periods were minimized. An audiogram (see Fig. 35, lower) one year later revealed not only improvement in the hearing in the operated ear, but also improvement in the hearing in the unoperated ear. It is probable that unsuspected vasomotor cochlear impairment had been present even before operation.

Comment: Otologists have frequently remarked improvement in the unoperated as well as in the fenestrated ear in certain otosclerotics following fenestration surgery. Some of

these may be cases of otosclerosis in unstable vasomotor-reacting individuals who, when the emotional strain associated with hearing impairment has been lightened by a good fenestration result, regain a more normal general vasomotor tonus which in terms of the cochlea may mean improved hearing — even in the unoperated ear.

In otosclerotics with associated inner ear impairment with high tone drop we have made it a habit to check thoroughly into the neurovascular background and give due attention to the historical fluctuations of acuity associated with weather change, fatigue, menstrual-cycle, or emotional tension. Periodic dependence upon vasodilating and blocking agents, vitamin concentrations, and hormones where purposeful have paid off in maintenance of our fenestration results.

CONCLUSIONS.

1. The labyrinth is one of the most discrete and symptomatic reactors in the field of peripheral vascular disorder.
2. The dead end of therapeutic nihilism no longer applies to that large segment of inner ear disorders resulting from peripheral vasomotor change.
3. By aggressive neurovascular stabilization a significant segment of imperiled inner ear function can be salvaged and maintained.
4. A considerable responsibility rests with otologists to teach the early recognition of these vasomotor disturbances, so aggressive therapy can be applied promptly.
5. The otologist cannot be a narrow surgical technician. He has also an important medical mission to perform.
6. Otology in this regard has a fascinating and an unlimited horizon.

CONGENITAL TRACHEOESOPHAGEAL FISTULA NOT ASSOCIATED WITH ATRESIA OF THE ESOPHAGUS.*

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INTRODUCTION.

Congenital tracheoesophageal fistula is one of the less common developmental anomalies, but it has received considerable attention during the last 15 years and is now one of those anomalies which can be successfully treated by surgery. The increasing interest in the diagnosis of all congenital defects has resulted in the earlier recognition of an increasing number of cases of tracheoesophageal fistula *with* the usually associated atresia of the esophagus, and by this earlier recognition more successful treatment has been accomplished. Mortality in such cases is steadily decreasing, whereas up to 1939 no patient had survived surgery. In 1940, Lanman[1] reported 32 cases studied at The Children's Hospital in Boston between 1928 and 1938. All but one of these children had the usually associated atresia of the esophagus. Thirty of these patients were operated upon, with a mortality for the series of 100 per cent. During the following eight-year period (1939-1947) at the same institution, Swenson,[2] in 1948, reported 113 such cases, 57 of whom survived surgery—a mortality of only 50 per cent.

The purpose of this paper is to point out that there is now evidence that there is an increasing number of cases in which

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congenital tracheoesophageal fistula is present *without* an associated atresia of one or both segments of the esophagus, although over 90 per cent of the cases of esophageal atresia reported in numerous series have shown a tracheoesophageal fistula. Up to now it has been assumed that congenital tracheoesophageal fistula existed only in conjunction with an esophageal atresia. As far as can be determined from the literature to date there are only two cases of surgical closure of a congenital tracheoesophageal fistula in which there was no associated atresia of the esophagus. In 1939, Imperatori[3] reported a case of a six and one-half-year-old boy in whom he closed a high tracheoesophageal fistula through a cervical tracheotomy incision. Two operations were necessary to complete the closure. Haight,[4] in 1948, reported the other case of closure of a simple tracheoesophageal fistula not complicated by esophageal atresia. This was in a four-year-old boy whose fistula was closed in one operation by an extrapleural thoracic approach. So far no case has been reported in which diagnosis was made and surgical treatment carried out during infancy. It is important to discuss in detail the difficulties in diagnosis of tracheoesophageal fistula without atresia, since such cases must be adequately recognized at an early stage of life if proper treatment is to be carried out, and if irreversible damage to an already infected pulmonary system is to be avoided. In the past the rarity of cases of tracheoesophageal fistula not complicated by esophageal atresia, plus the less striking symptoms in the earlier stage of life, are reasons that this unusual type of anomaly has been overlooked.

The conclusion that the passage of a catheter into the stomach or that the swallow or flow of a radio-opaque contrast medium down the esophagus is sufficient to rule out tracheoesophageal fistula is a mistaken one and must be corrected. It will eliminate the possibility of only an atresia. It is particularly important for otolaryngologists to realize that such an anomaly without either atresia or stenosis of the esophagus may exist, and to employ improved methods of diagnosis when the simple passage of a catheter or the use of a fluoroscope

fails to reveal evidence of esophageal obstruction, yet the patient's symptoms point to digestive or respiratory tract difficulties.

Included in this study are the reports of five hitherto unreported cases of infants with congenital tracheoesophageal fistula seen at The Children's Hospital in Boston during the past four years. None of these patients had an associated atresia of the esophagus. Two infants were successfully operated upon and the fistula closed. They are now living normal lives. One baby died postoperatively, and the results of the postmortem examination are also presented. The fourth baby was not operated upon but the tracheoesophageal fistula was satisfactorily demonstrated clinically. The fifth infant was dead on admission to the hospital, but the postmortem findings of this patient are presented.

HISTORICAL REVIEW.

Durston, in 1670, is generally credited with having reported the first case of congenital tracheoesophageal fistula, and it was associated with the usual atresia of the esophagus (quoted by Ladd[5] and Mackenzie[6]). In "Anatomy of Human Bodies Epitomized," in 1703, Thomas Gibson[7] described the clinical and pathological findings of a patient whom he had observed in 1696. His original description is most interesting:

"About November, 1696, I was sent for to see an infant that could not swallow. The child seemed very desirous of food and took what was offered it in a spoon with greediness, but when it went to swallow it, it was like to be choaked; and what should have gone down, returned by the mouth and nose, and it fell into a struggling convulsive sort of fit. It was very fleshy and large and was two days old when I was called to it, but the next day died.

"The parents being willing to have it opened, I took two physicians and a surgeon with me. . . . We cut open the thorax and taking out the gullet continued to the stomach. Then we made a slit in the stomach, and put a pipe in its upper orifice,

and blowing we found the wind had a vent, but not by the top of the gullet. Then we carefully slit up the backside of the gullet from the stomach upwards, and when we had gone a little above halfway toward the pharynx, we found it hollow no further. Then we began to slit it open from the pharynx downward, and it was hollow til within an inch of the other slit, and in the imperforate part it was narrower than in the hollowed. The isthmus (as it were) did not seem ever to have been hollow, for in the bottom of the upper and the top of the lower cavity there was not the least print of any such thing, but the parts were here as smooth as the bottom of an acorn cup. Then searching which way the wind had passed when we blew from the stomach upwards, we found an oval hole (half an inch long) on the fore side of the gullet opening into the aspera arteria (lower trachea), a little above its first division, just below the lower part of the isthmus above mentioned."

Thus there is no question but that over 250 years ago Gibson had made a most accurate observation of a congenital tracheoesophageal fistula with the commonest of associated anomalies (esophageal atresia), in which the fistulous tract connects the trachea and the lower esophageal segment (Type III b. of Vogt[8]).

In 1873, Lamb[9] is credited with having been the first in North America to report a case of tracheoesophageal fistula without other esophageal abnormalities. The baby died at the age of seven weeks. In 1884, Mackenzie,[6] going back to 1670, collected 62 cases of congenital atresia of the esophagus, 43 of which had a tracheoesophageal fistula, and added one of his own. In 1917, Cautley[10] merely listed from the literature six cases of tracheoesophageal fistula supposedly without atresia, and Lamb's case[9] was one of them. He did not discuss the cases. In a very complete review of the literature up to 1919, Plass[11] reported 136 cases of the anomaly, all but one of which were associated with an esophageal atresia, and that one was the case of Lamb noted above. Haight[4] has taken the time to verify these six cases listed by Cautley. In addition to that of

Lamb there were five others. Eppinger's case was that of a congenital tracheoesophageal fistula just above the bifurcation of the trachea and a normal esophagus. The second case, reported by Vrolik, was considered by Plass as having an associated atresia, but according to Eppinger had a narrow esophageal stricture below the fistula. The patient described independently by Pinard and Tarnier was also considered by Plass to have an atresia, but Haight's consultation with the original reference determined that he had a high tracheoesophageal fistula just below the glottis and no atresia of the esophagus. The cases of Richter and von der Water could not be verified by consultation with the original papers.

Vogt,[8] in 1929, offered the first attempt at classification of tracheoesophageal fistula and congenital atresia of the esophagus, but did not mention the possibility of tracheoesophageal fistula occurring without atresia.

Type I. Complete absence of the esophagus. (Very rare type.)

Type II. Atresia of the esophagus with separate upper and lower segments, each ending in a blind pouch.

Type III. Atresia of the esophagus with a tracheoesophageal fistula:

a. Fistula occurring between the upper segment of the esophagus and the trachea.

b. Fistula occurring between the lower segment of the esophagus and the trachea (Commonest type.)

c. Fistula arising in both segments of the esophagus and connecting with the trachea.

Ladd[5] has added a Type IV (a variant of Type III c of Vogt) in which there is an atresia of the esophagus with a fistula which arises at the bifurcation of the trachea instead of above it and enters the lower segment of the esophagus. One of Lanman's[1] cases (listed as Type III c) showed an upper esophageal segment with a tiny tracheoesophageal fis-

tula as its only outlet, and a lower segment which also opened into the trachea at the same level. In this case the only communication between the upper and lower esophageal segments was a detour via the trachea (Type V of Ladd). Holt, Haight, and Hodges[12] have suggested adding another classification to include the subject under discussion—namely, tracheoesophageal fistula *without* atresia (see Figs. 1 and 2). In 1929,

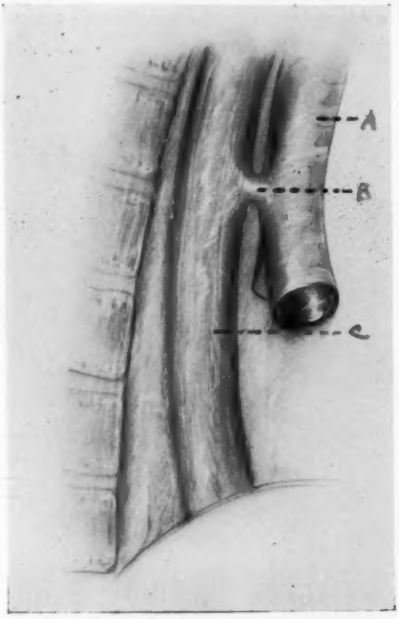

Fig. 1. Schematic drawing of a tracheoesophageal fistula without atresia of the esophagus. (A) Trachea. (B) Fistula. (C) Esophagus. From Swenson (2) (p. 199).

Negus[13] reported the case of a 45-year-old man who died of carcinoma of the lung. Postmortem examination showed a congenital opening running upward from the esophagus into the trachea. No symptoms had been noted which could have been attributed to the fistula. This was felt to be due to the fact that a small valve-like hood of mucous membrane was

found on the esophageal aspect of the fistula. This flap may have temporarily closed the opening during swallowing and prevented the regurgitation of food into the trachea. In 1931, Rosenthal[14] in an excellent and comprehensive paper described both the gross and microscopic pathology found in cases of tracheoesophageal fistula. He reported 205 congenital tracheoesophageal fistulae occurring in a total of 255 cases of atresia

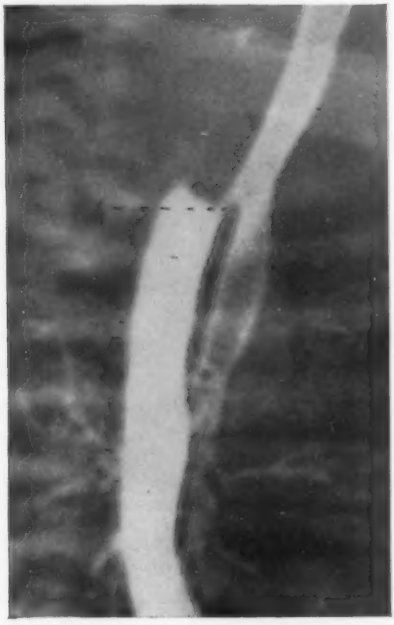

Fig. 2. Oblique Roentgenogram of intratracheal injection of lipiodol showing tracheoesophageal fistula (A) with no atresia of the esophagus.

of the esophagus, including eight of his own. Of this total group of 255 cases, 205 had the commonly associated fistula, 40 did not have a fistula, and 10 were not autopsied so its presence or absence was not ascertained. Rosenthal makes no mention of fistula without atresia of the esophagus. In 1933, McKinney[15] reported a patient whom he suspected had a con-

genital tracheoesophageal fistula. This was a 58-year-old woman who had for 25 years complained of gagging, vomiting, and a paroxysmal cough associated with and also following meals. A gastrointestinal series of Roentgenograms showed barium in the right bronchial tree. Although esophagoscopy was negative, bronchoscopy showed what was described as a "probable" opening in the tracheal wall from which a creamy fluid was emanating; however, this cannot be regarded as a proven case of congenital fistula.

It was not until 1939 that a successful cure of a case of tracheoesophageal fistula was first accomplished. In that year both Ladd and Leven successfully operated upon the first patients with congenital tracheoesophageal fistula associated *with* atresia of the esophagus. Leven[16] reported his case in 1941. Ladd's[5] case was 24 hours older but was not reported until 1944. In 1939, also, Imperatori[3] reported the first attempt to make a surgical closure of a congenital tracheoesophageal fistula *without* an associated atresia. This was an interesting case of a six and one-half-year-old boy who had been treated for years as a difficult feeding problem. It was thought that he probably had a stricture of the esophagus so the esophagus was dilated several times. Regurgitation of food continued, and the patient went through many bouts of bronchopneumonia. While swallowing a string in preparation for one of the dilatations of the esophagus, the string suddenly went through a congenital fistula into the trachea and produced marked respiratory difficulty. Emergency tracheotomy was necessary. When the patient was six years old, Dr. Chevalier L. Jackson demonstrated bronchoscopically a slit-like fistula 1.3 cm. in length in the region of the third, fourth and fifth tracheal rings. Imperatori closed this fistula in two operations through a cervical tracheotomy incision and the patient thenceforth did most satisfactorily. In 1940, Lanman,[1] in a very thorough paper, reviewed the literature and presented 31 cases of fistula in 32 patients with esophageal atresia treated at The Children's Hospital in Boston. In 1941, Gengenbach and Dobos[17] stated that although tracheoesophageal fistula without atresia exists, such conditions are obviously

quite rare. They did not mention Imperatori's case, but reported two patients of their own who were proven to have congenital tracheoesophageal fistula without atresia, and one in whom the possibility was suspected clinically but never proven. The first was a three months old baby girl who had had a history of repeated respiratory difficulty associated with attacks of cyanosis since birth. She had regurgitated many of her feedings and cyanosis was associated with feeding. Barium studies showed no esophageal obstruction, but the barium entered the trachea. This was suspected to be due to a faulty deglutition mechanism of the epiglottis. Following the ingestion of the barium mixture the baby developed a severe bronchopneumonia and died. Autopsy, however, showed a small tracheoesophageal fistula 4 mm. in diameter located 3 mm. above the bifurcation of the trachea.

The second patient was a ten months old baby girl who since the third day of life had experienced attacks of cyanosis and coughing while feeding. She had been cyanotic at birth and also had had repeated attacks of bronchopneumonia, Bronchoscopy by Dr. Holinger in Chicago showed a small orifice 2 to 3 cm. above the bifurcation of the trachea, through which clear frothy fluid exuded. An apparent esophageal obstruction was encountered 19 cm. from the teeth. There is no record of therapy in this case, and since she was still living at the age of two years she obviously did not have esophageal atresia.

The third patient was a two and one-half months old female child who had the same type of history with a persistent cough since three weeks of age, when she began to have choking and cyanotic attacks associated with feedings. Esophageal lipiodol injection was performed, following which fluoroscopic and film studies were made. On the films lipiodol was seen in the left main bronchus and trachea. The diagnosis was made only on this evidence, as no definite fistula could be demonstrated. Neither esophagoscopy nor bronchoscopy was permitted, and a fistula was merely suspected.

In 1945, Leven and Lannin,[16] in a discussion of 41 cases from the University of Minnesota Hospitals, mentioned two patients who had a tracheoesophageal fistula without atresia. Three cases in this series had a simple atresia without fistula, and the other 36 the usual combination of fistula with atresia. They state, however, that diagnosis before autopsy is extremely difficult because the fistula may be small, symptoms inconstant and the diagnosis open to question.

Although nearly 100 patients with esophageal atresia had been seen at The Children's Hospital in Boston up to 1946, none had been diagnosed as a congenital fistula unassociated with an atresia of the esophagus.

The fifth case in this present series is an autopsy report of that first patient with a fistula without atresia.

In 1946, Holt, Haight and Hodges,[12] in a series of 46 cases from the University Hospital in Ann Arbor, Mich., mentioned one case of congenital tracheoesophageal fistula without atresia. In 1948, Haight[4] reported the second case which he had seen at the University Hospital and is now credited with having reported the second successful operation on a congenital fistula without atresia. The only two cases reported in which the anomaly was recognized by endoscopic methods were those of Imperatori[3] (bronchoscoped by Jackson) and Gengenbach and Dobos[17] (examined by Holinger).

INCIDENCE.

In a discussion on the incidence of tracheoesophageal fistula without atresia it is impossible to obtain accurate information because of the paucity of cases reported. In the past it was nearly always considered that the fistula occurred in conjunction with an atresia of the esophagus. Many cases of fistula with an otherwise normal esophagus must have been missed, and the incidence, therefore, may be much higher than one would suspect even at the present time. With more widespread knowledge that such anomalies do occur, and with better diagnostic methods employed in patients with vague gastrointestinal and respiratory complaints, the incidence may

be found to be even higher in the future. For the present, however, the incidence will have to be considred on the basis of cases with associated esophageal atresia. In 1948, Haight[4] stated that during the preceding 12 years 63 infants with congenital atresia of the esophagus had been seen at the University of Michigan Hospital and that of these, only two had a fistula in the presence of an otherwise normal esophagus.

In an excellent article on incidence and possible etiology of this condition, Ingalls and Prindle[19] reported a study of 107 cases of infants with esophageal atresia, 102 of whom had an associated tracheoesophageal fistula. All of these patients were seen at The Children's Hospital in Boston from 1936 to 1948 or were born at the Boston-Lying-In Hospital between 1934 and 1945. They found that six cases had occurred at the Boston Lying-In Hospital during this period where 30,497 live births were reported (one was stillborn). On this basis they estimated the frequency of tracheoesophageal fistula in the Boston area as at least 0.2/1,000 live births, the major variable being the missed case (stillborn) and asphyxial death in which no autopsy is performed. They quote Murphy, who reported, in 1947, 13 cases in 166,451 live births in Philadelphia in a five-year period—an incidence of less than one in 10,000. A study of the literature revealed no case of true tracheoesophageal fistula which recurred as a familial trait in successive generations. Mackenzie,[6] however, reported the condition in three children of the same generation fathered by one man but born of different mothers. Lanman's[11] series of 32 patients included in the same family two children with tracheoesophageal fistula and esophageal atresia (Type III b of Vogt[8]). One was a boy born in 1932 and the second a girl born the following year. In 1939, Grieve and McDermott[20] reported two cases in successive male births in the same family in 1937 and 1938. Both of these babies died and postmortem examinations were performed. The fistula entered the lower esophageal segment (Type III b). An interesting feature is that the mother and father were third cousins. Ingalls and Prindle[19] state that in their series the condition occurred more often in males than in females, with a ratio of 62:45;

however, on adding cases from the literature to those of their own, the incidence showed only 57.6 per cent in favor of the males, and this slight difference was not interpreted as significant on a statistical basis. These authors state that such babies are often born to mothers in the older age group, although such tendency is not so striking as is seen in Mongolism. They did notice a higher incidence, however, in unmarried mothers and brought up the possibility that attempted abortion may have affected the developing embryo. It is interesting to note that many authors such as Plass,[11] Ballantyne,[21] and Ingalls and Prindle[19] as well, have wondered about the higher incidence of cases with associated hydramnios or anomalies of the placenta. Of 87 mothers in the Ingalls and Prindle series, 20 showed hydramnios. They feel that unquestionably placental disease can affect the nutrition and oxygenation of the fetus, so, therefore, it might produce structural abnormalities as well.

ETIOLOGY AND EMBRYOLOGY.

In a detailed consideration of any congenital anomaly a study of its embryological background is the only means by which additional light might be thrown upon possible causative factors. A brief review of the local embryology is, therefore, in order.

The trachea and esophagus develop from the cephalic portion of the primitive foregut. The upper (or paratracheal) portion develops from the retropharyngeal segment of this foregut, while the lower section differentiates from its pregastric segment—thereby indicating a separate development of both upper and lower esophageal sections. A single tube is formed. As early as the 3 mm. stage of the embryo the normal pulmonary anlage originates from the ventral portion of the primitive foregut just caudal to the pharyngeal pouches as a small mass of entodermal cells. By the 4 mm. stage this mass of cells has grown caudally so that there are formed externally along the foregut wall two lateral longitudinal grooves. Internally these grooves form corresponding elevations which eventually produce a septum which divides the foregut into

two completely separate hollow tubes, — the anterior trachea and the posterior esophagus. On the ventral surface of the newly formed trachea develop the lung buds. These changes occur during the second fetal month so that at the end of seven or eight weeks of intrauterine life the trachea and esophagus should have been completely separated and normally developed. By the time the embryo has reached the 11 mm. state it should either have a normal trachea and esophagus, or the anomaly under discussion will have been established. The cause of such an anomaly as a persistence of an opening from one of these tubes to the other (*i.e.*, tracheoesophageal fistula) or a complete obliteration of the lumen of the posterior tube (*i.e.*, esophageal atresia), is not clear. The defect is usually very constant and must be present very early in embryonic life. An arrest in development of the septum could produce an incomplete separation of the two tubes and result in a fistula. Cause for this arrest in one particular area, however, is the crux of the problem, but the answer is by no means clear. This arrest has more generally been regarded as the result of a primary deficiency in the developmental capacity of the entodermal cells in the lateral masses rather than the result of some extraneous factor. The occurrence of the same defect in both halves of Ysander's[22] monster embryo would tend to substaniate this thesis. In 1924, Ysander described an 8 mm. monster embryo of the type Thoracopagus tetrabranchius in which there was but one stomach and one heart anlage while the esophagus and respiratory apparatus were duplicated identically. Each "embryo" presented a typical tracheoesophageal fistula with associated atresia such as is seen in full-term babies. This is the youngest embryo showing such an anomaly. Gruenwald,[23] in 1940, reported the condition in a 9 mm. embryo of about the same relative age. Minot[24] reported an 18.1 mm. embryo in the Harvard Embryological Collection which had a similar deformity. The association of other congenital malformations, especially those of the gastrointestinal tract such as the commonly associated imperforate anus, or other atresias, makes one strongly consider an inherent defect of cellular development.

In a discussion of possible causes for such maldevelopment, Rosenthal[14] refutes all theories except that of altered cell growth along the lateral ridges. This defective or absent growth of the septa would result in a fistula, whereas deficient growth of entodermal cells in the dorsal wall of the foregut would result in atresia of the esophagus.

It had been considered by Kreuter[25] that esophageal atresia was the result of cessation or retardation of growth of the esophagus at a stage when the tube had become a solid bar of tissue with no lumen. This condition supposedly is produced by proliferation and concrescence of the epithelial lining of the esophageal tube. Later on, in the normal course of events theoretically the lumen is re-established by vacuolization followed by coalescence of the vacuoles; however, Rosenthal considers this theory untenable because Förssner[26] demonstrated that the "solid bar" stage does not appear until the 20 mm. embryo, yet the tracheoesophageal anlage is well formed and the trachea is completely separated from the esophagus normally by the 4 to 6 mm. stage. The finding of the anomaly in these young embryos under 18 mm. (described by Ysander, Gruenwald and Minot) is concrete evidence for the refutation of this theory. In adddition there is apparently some question as to whether the above described process of obliteration of the lumen into the "solid bar" stage with eventual vacuolization and re-establishment of the lumen occurs in the process of normal development of the embryo. Schridde[27] found in an extensive study of 50 human embryos a normal lumen in the esophagus at all stages of development.

Keith and Spicer,[28] in a report of three cases of malformation of the tracheoesophageal septum with atresia and fistula noted the association of a deep origin of the right subclavian artery crossing from the aorta to the right side between the two esophageal segments. Such findings give credence to the theory that abnormal pressure of such vessels or of an abnormally developing heart might result in the separation of the continunity of the esophagus. Rosenthal,[14] however, believes that such cardiac or vascular anomalies are the effects rather

than the causes of the development of esophageal atresia. The esophageal defect is already present, so during the process of development of the vascular tree these early vessels come to lie in the most easily accessible positions—namely, in hollow spaces in which there are no other organs, as in the area between the two pouches of the separated esophagus. In Cases 3 and 4 of the present series it is quite interesting that on fluoroscopic examination by lipiodol swallow a posterior esophageal defect was seen running obliquely upward from left to right at the level of the second or third thoracic vertebrae. This was interpreted as the characteristic appearance of an aberrant subclavian artery. At the time of operation in Case 3 this artery was readily identified as such, and was ligated and divided before the fistula was closed. Case 4 of the present series also presented the Roentgenologic appearance of an aberrant right subclavian artery. Gengenbach and Dobos[17] also suggested that the most plausible theory for this abnormal development was one advocated by Zausch (*Arch. f. Path. Anat.*, 239:94:1921), that pressure on the primitive entodermal sac by the enlarging heart anlage was sufficient to cause the anomaly. Rosenthal,[14] however, states that such pressure is greater on the trachea than on the esophagus, yet it is the latter structure which shows atresia. He also feels that there is an adequate space between the developing heart and tracheoesophageal anlage. In the paper by Ingalls and Prindle,[19] Dr. Frederick T. Lewis, of the Harvard Medical School, discusses a possible explanation along the lines of abnormal pressure in the developing young embryo. He quotes a monograph by Broman on the development of the omental bursa:

"In human embryos of 3.4 mm. the common cleft-like cavity that later becomes vertically subdivided into the ventral trachea and the dorsal esophagus is closely flanked on either side by similar extensions of the peritoneal cavities. These extensions Broman named significantly the right and left 'pneumatoenteric recesses' but without recognizing their possible relation to fistula and atresia. Any fluid that they contain would be separated from that within the amnion and within the yolk-sac, but at that stage it would not be separated from

subplacental fluid within the chorion. By connection with the pericardial cavity the contents of the recesses might be subject to pressure changes as the heart filled and emptied. Compression of the foregut between the recesses could divide the esophagus along their dorsal borders and produce the characteristic anomaly under discussion. They change in form rapidly as the left one disappears and the right gives rise to the superior recess of the omental bursa. In an older embryo which still measured only 3 mm., Broman has shown the anterior ends of these recesses crossing the esophagus and yet already below the site of the anomaly. An embryo of 4 mm. figured as a whole by Bremer who clearly reconstructed most of its organs was used to show how the associated atresia and fistula presumably arise. Malformation of the pneumatoenteric recesses perhaps with undue pressure from their contents in a brief period toward the close of the first month could produce the anomaly. At 8 mm. it should be well established."

Dr. George L. Streeter, quoted by Rosenthal[14] in a personal communication, suggested the possibility that tracheoesophageal fistula was produced in the same manner as spina bifida and presented this simple analogy. In the stage when the neural tube is open, closure takes place not by mesenchyme pushing in the entoderm, but by cellular proliferation which later results in fusion. If there is a deficiency in the cells resulting in altered proliferation and nonunion, the tube remains open (Spina bifida). In the tracheoesophageal situation the two tubes, a dorsal esophagus and ventral trachea, normally are formed from one original tube as has been outlined. If for any reason a deficiency in the cells which should form the septum causes altered proliferation and nonunion, a tracheoesophageal fistula persists. The deficiency in the dorsal entodermal cells that were to give rise to the esophagus directly above this area may result in its nonformation (*i.e.*, atresia). In the very early embryo (presomite stage) the anterior termination of the neurenteric canal is in direct proximity to that portion of entoderm where defective growth occurs. There may, therefore, be some correlation between

the establishment of spina bifida and tracheoesophageal fistula. Mackenzie's[6] story of three children of the same father but of different mothers very strongly suggests an inherent genetic factor which may be responsible for the altered cellular growth in the local area described above.

Trauma and intrauterine inflammation or infection have always been propounded as possible causes of this as well as of many other congenital anomalies (*e.g.*, Luschka and Klebs, cited by Rosenthal[14]). In a paper in 1947 on epidemiological implications of developmental arrest, Ingalls and Gordon[29] discussed agents which could be regarded as possible causes of prenatal disease. They divided such agents into three categories: namely, *1. Infectious* (rubella, syphilis, etc.); *2. Chemical* (nutritional deficiencies, toxic and metabolic disturbances, etc.); and *3. Physical* (mechanical trauma, structural disease of the uterus, etc.). It is now generally accepted that an infectious agent may produce changes in the developing embryo of humans, as has been well known for some time in other animals, and that often these changes have a constant pattern from animal to animal. During the past several years since the studies of an epidemic of rubella in Australia it has become common knowledge that such an infectious agent as this particular virus when contracted by a mother in her first trimester of pregnancy very often produces permanent structural changes in the developing embryo. These changes are in the nature of congenital malformations which run a rather constant pattern: namely, the association of congenital cataracts; total deafness on the basis of VIIIth nerve or cochlear malformations; microcephaly; and various cardiac defects. All of these organs affected are in the process of maximum embryonic development during the first trimester of pregnancy. Mothers contracting rubella after the first trimester of pregnancy do not tend to produce infants with such defects. In Mongolism, Ingalls has postulated that the specific clinical defect produced is a nonspecific function of the stage of embryonic development, and not a specific property of the individual agent. Ingalls and Prindle,[19] therefore, conclude that it is possible that congenital tracheoesophageal fistula

and certain commonly associated defects of the cardiovascular, gastrointestinal, genitourinary, respiratory and skeletal systems may represent departures from normal sequences of development at about the fifth or sixth week of embryonic life caused by agents acting through the mother and the placenta. Significant is the association of 20 cases of hydramnios occurring in the total group of 87 mothers of infants with such malformations of the esophagus. In this group four mothers had twins. In six instances antepartum hemorrhage occurred (five of these were during the first or second months of pregnancy). Six of the mothers suffered from acute infections or metabolic disturbances in the first trimester of pregnancy. These conditions were listed as acute upper respiratory infections, pyelitis, appendicitis and pneumonia. A possible metabolic factor in one patient was the ingestion of 30 gm. of quinine for an attempted abortion.

Ballantyne,[21] Brigham,[30] Meyer[31] and Scheurer[32] noted an increased incidence of hydramnios associated with these anomalies and believed it to be the result of the inability of the fetus to swallow and absorb amniotic fluid. They considered the usually noted dilatation and hypertrophy of the upper esophageal pouch in cases of atresia the result of ineffectual attempts on the part of the fetus to swallow this fluid.

Strong and Cummins[33] give credence to the theory of both genetic and environmental factors by stating that the agents predisposing to this anomaly are to be sought either in the reproductive cells from which the child is produced, or in an unfavorable intrauterine environment existing during the critical early period of embryonic life.

In a discussion of arteriovenous fistula, Reinhoff[34] states that there is in the life cycle of a cell or tissue which is differentiating or growing, a period in which that cell is very sensitive to its environment, and any injury to it at that stage will result in the abnormal growth and development of that cell or structure.

The problem, therefore, still exists and, so far there has been nothing but speculation as to possible causes of this, as

well as other, congenital anomalies. It is quite possible that some such extraneous factor as Ingalls and Prindle have mentioned may so affect the development of certain organs that malformations are produced. It is by no means clear, however, why these influences should affect only certain groups of cells and produce only certain constant patterns of anomalies and at only a certain time during pregnancy. A genetic factor influencing particular groups of cells cannot be excluded.

PATHOLOGY.

The best description of the pathological anatomy and histology of the condition of congenital tracheoesophageal fistula and esophageal atresia is by Rosenthal.[14] He made a careful gross study of eight cases and in four of these examined the tissues microscopically. In these eight cases he concluded that the average site of the fistula above the bifurcation of the trachea was 0.5 cm. In two cases it was at the bifurcation (Type IV of Ladd[5]); in one, just above the bifurcation; in two cases, 0.5 cm. above, and in the three others, 1 cm., 1.5 cm. and 2 cm. above the bifurcation, respectively. These findings confirm the reported figures of Plass[11] in his review of the literature, and of many others. In Plass' series of 96 cases, 94 fell into this same small range, while in two cases the fistulous tract extended from a bronchus into the esophagus. In 80 later cases mentioned by Rosenthal, four showed a fistula between the esophagus and the right main bronchus and one between the esophagus and left main bronchus.

In general, the fistula has a crescentic outline and has been described as similar to the appearance of the ureteral orifice in the bladder. The edge of the fistula is smooth and regular with a continuous mucous membrane covering the trachea, esophagus, and its communicating tract. The muscular walls frequently blend with each other and have no definite line of demarcation. In the posterior portion of the membranous trachea there may be noted a groove-like depression running upwards from the fistula. In the fistulous portion of the esophagus Plass[11] reports that rudimentary tracheal cartilages

have been found. Rosenthal[14] quotes the work of Ladwig, who reported a case in which the lower portion of the esophagus continued into the posterior tracheal wall, and thick circular and longitudinal muscle layers and esophageal glands were found in the trachea itself. In the posterior tracheal wall stratified squamous epithelium extended up as far as the thyroid cartilage. Ladwig considered the posterior tracheal wall in such cases to be in reality an esophageal structure rather than a tracheal structure. Konopacki (from Rosenthal[14]) also found squamous epithelial cells in the tracheal mucosa in the region of the fistula and an abnormally thick muscular layer of the trachea where there were many cross-striated fibres. These findings were also corroborated by Gutmann[14] who noted that the muscle fibres of the posterior esophageal wall continued into the anterior tracheal wall as well. Rosenthal was the first to note tracheal structures in the lower portion of the esophagus. In three of his four cases studied histologically, the mucosa of the lower esophagus near the fistula was of the tracheal type with pseudostratified columnar epithelium, and in all four cases glands were found scattered through the muscle fibres of the esophagus. Respiratory epithelium in the esophagus, however, has been reported before in the case of normal newborn infants.[35]

The location and size of the fistula, as well as the adherence of the trachea and esophagus, have a bearing upon the surgical treatment of the fistula and upon the difficulties expected to be encountered.

SYMPTOMS AND DIAGNOSIS.

With such marked improvement in the surgical techniques which have brought about a steadily decreasing mortality in cases of congenital tracheoesophageal fistula or atresia of the esophagus, such cases must be diagnosed early so that proper surgical treatment can be instituted. In addition, it must be borne in mind that congenital tracheaesophageal fistula does exist without an associated esophageal atresia, and that in such cases the symptomatology may not be as striking as in the usually encountered anomaly which has an associated

atresia. In the presence of vague respiratory and digestive complaints this condition must be suspected until the possibility of its existence is adequately eliminated. Often there may be merely a history of several admissions to the hospital for recurring bouts of acute pneumonitis or bronchopneumonia. Perhaps the story may be even less striking and the patient is considered simply to be unusually susceptible to otherwise insignificant respiratory infections. In many patients the persistence of a loose chronic cough may result in the diagnosis of chronic bronchitis and no further attempts are made to evaluate the situation. With greatly increased use of bronchoscopy and lipiodol studies in infants, many cases of recurrent bronchopneumonia are now found to be due to congenital anomalies of the tracheobronchial tree, such as stenosis of a bronchus, abnormal origin or abnormal twisting of a bronchial segment, or partial agenesis of the tracheobronchial tree or pulmonary system. Reliance in the simple elimination of a stenosis or atresia of the esophagus is dangerous, as the patient still may have a fistula.

In many cases of congenital tracheoesophageal fistula, if the opening is sufficiently small, the baby may be able to nurse properly and take his feedings satisfactorily. He may show a steady weight gain with no abnormal symptoms. In other cases, attacks of choking while swallowing may occur with or without cyanosis. Very often such patients are able to take solid or semisolid food with much more ease than liquids. In such patients the possibility of a congenital tracheoesophageal fistula should be strongly considered. Attacks of choking or cyanosis may occur during a feeding or sometimes at a considerable time after feeding, especially if the infant is placed in a prone position as so often is customary. In such a position he tends to regurgitate feedings which by gravity pass anteriorly through the fistula into the tracheobronchial tree. An increased amount of air in the stomach and intestines may lead to suspicion of a tracheoesophageal fistula. Inasmuch as infants normally have a considerable amount of air in the gastrointestinal tract as most of this air is swallowed during feeding, it becomes difficult to decide whether the amount is

or is not abnormal; however, in cases of fistula, by closing the epiglottis as occurs during crying, vomiting, or straining, pressure in the tracheobronchial tree is thereby increased and air rapidly passes posteriorly through the communication into the esophagus and thence dilates the stomach. The cricopharyngeus muscle must also close spontaneously at this time so that the air is not passed to the outside through the pharynx and mouth, but is forced downward into the stomach and intestine, with resultant marked distention.

There is generally an increased amount of mucus in the mouth, pharynx and nasopharynx of such an infant, and very often the mucus may have a frothy appearance. A persistent loose, rattling type of cough occurs, and vomiting while coughing is frequently noted. The mucus, however, is not so troublesome as in patients with associated atresia of the esophagus, where it is necessary to employ constant suction to prevent further aspiration into the tracheobronchial tree.

Clinical examination of the patient may show good nutrition and other wise normal development, as most of these babies appear normal at birth, and can maintain adequate growth if the repeated respiratory infections are properly controlled. This is much easier today with the extensive use of chemotherapy and antibiotics. Often, however, other serious congenital defects will manifest themselves. The chest usually shows coarse rales and rhonchi throughout both lung fields, but very often these signs are more localized to the right upper lobe. Examination for tactile fremitus indicates an increased amount of mucus in the entire pulmonary system. The abdomen may be distended and tympanitic especially after crying, straining or even vomiting.

In the presence of choking and cyanosis during feedings, passage of a catheter from the mouth to the stomach should be the first diagnostic procedure in the case of a newborn infant. If an atresia is found, the obstruction is usually met when the catheter is 10 to 12 cm. from the alveolar ridge and its position with the fluoroscope is seen at the level of the second thoracic vertebra. If, however, no difficulty is encountered

in the passage of the catheter, examination obviously must not stop here, especially if symptoms continue. The next step is the use of some radio-opaque contrast medium swallowed or injected via the esophageal catheter. Barium should never be used in such a situation, as aspiration or leakage through a possible fistula will result in pulmonary changes which may well become permanent. It hardly seems necessary to call attention to the danger of aspiration of barium, but on numerous occasions it had been considered safe after the possibility of atresia of the esophagus had been eliminated. Lipiodol and iodochloral seem to be the best contrast media as both are nonirritating to the tracheobronchial tree and most of it is easily removed by suction. A very small amount (2 to 3 cc.) will usually suffice. Such an examination should be carried out under the fluoroscope as the fistula may be very small and often easily missed on a simple Roentgenogram. It is especially important, as Haight[4] has suggested, that the patient be kept in the prone position while the lipiodol is flowing down the esophagus. Otherwise, none of the oil may pass anteriorly through the fistula into the trachea. Holt, Hodges and Haight[12] discuss one case of fistula that had been missed Roentgenologically because the patient was examined with iodized oil only while in the supine position. This patient with congenital esophageal atresia developed a recurrence of the fistula after it had been ligated. It could not be identified nine or 10 months postoperatively on two fluoroscopic examinations while the patient was in the usual supine position. Because of the recurring or persistent pneumonitis, patency of the fistula was still suspected, and on the third examination it was immediately evident when an additional swallow of lipiodol was given with the baby lying on his abdomen. Under the fluoroscope patients with congenital tracheoesophageal fistula show a gastrointestinal tract which contains in most instances an unusually large amount of air. Spot films are taken for later study as it is quite possible that under the fluoroscope the fistula may be missed, yet subsequent careful study of the films will reveal only a peculiar "tent-like" appearance of the esophagus in the region of a fistula as noted at A-Fig. 3.

Once a fistula is suspected, examination is not complete until bronchoscopic and esophagoscopic studies have been performed. These procedures should be used much more commonly in the infant age group than is generally the case. There

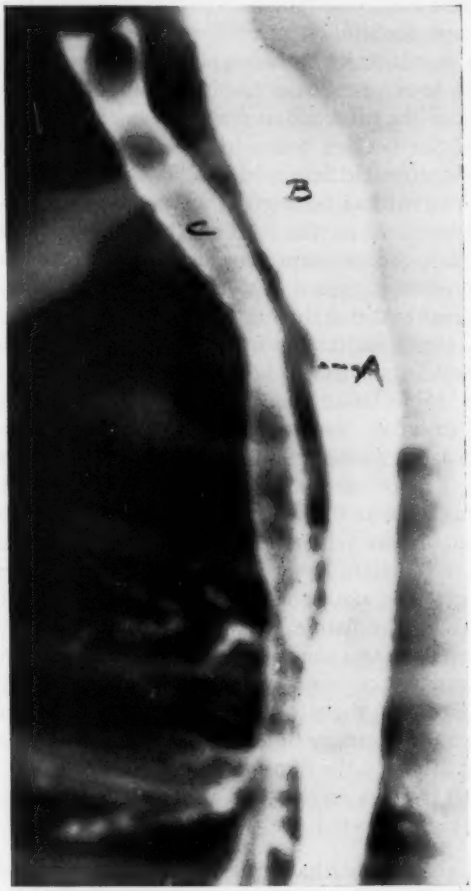

Fig. 3. Lateral lipiodol Roentgenogram showing "tent-like" defect on anterior esophageal wall (A) at site of fistula. (B) Esophagus. (C) Trachea. (From Swenson (2) (p. 199).

is no contraindication to the use of these methods even in newborn infants, and such procedures at this age are very frequently necessary. It is, to be sure, a somewhat more difficult problem when it is necessary to use a 3 or 3.5 mm. bronchoscope and a small fistula may be very difficult to visualize through such a narrow lumen. The use of a Broyles telescopic bronchoscope simplifies the problem as direct visualization of the fistula can thereby be accomplished. More careful inspection of the lower posterior tracheal wall in the region from 1.5 cm. above the bifurcation downwards should be performed as most of the fistulae occur in this locality. In the demonstration of traumatic fistula in adults, Abbott[36] has found that when the patient has been given a swallow of methylene blue prior to bronchoscopy the fistulous tract may be more easily seen by tracheoscopic examination as the dye leaks through the communication. Such a solution can be injected through an esophageal catheter during the bronchoscopic procedure in an infant, and visualization of the fistula will be much easier. It is preferable to shift the baby carefully into the prone position while this is being done so that dye will run through the fistula by gravity. Esophagoscopy should be carried out in undiagnosed yet suspected cases, although bronchoscopic examination is much more apt to result in better visualization of the fistulous tract as the tracheal lumen does not undergo such wide or constant movements as the esophagus, and the tracheal rings add to the stability of the wall. In Haight's[4] case of the four-year-old boy, examination with the 5 mm. esophagoscope failed to show the fistula. It was also impossible to see the opening from the tracheal side through a 4 mm. bronchoscope. When a 5 mm. bronchoscope was employed, a large fistula was identified although the site of the fistula had not been localized by fluoroscopic examination with lipiodol injection nor by subsequent study of the films. The oil, however, had been seen to enter the trachea on the first swallow, so the diagnosis had been strongly suspected.

A technique which I have previously used to outline a fistulous tract between the esophagus and a bronchus is of service in the diagnosis of a congenital tracheoesophageal fistula not

apparent by routine methods. A thin latex rubber balloon attached to a small, tightly ligated catheter is passed through an esophagoscope into the lower portion of the esophagus. The balloon is then inflated to close the esophageal lumen. Another similar balloon, which in addition has a second catheter passing either through or along the outside of the balloon (so that it projects just beyond the balloon's distal limit) is inserted into the very upper portion of the esophagus. The central portion of the esophagus can thereby be distended by the injection of air through the second or uppermost catheter into the closed esophageal segment. A small amount of lipiodol and air is then injected through the catheter and the increased pressure forces the lipiodol into the trachea via the fistula. Very little pressure is necessary. The fistula can be well outlined, and the troublesome question of reflux up the esophagus and aspiration of lipiodol through the glottis is eliminated. This is of considerable value in the sometimes troublesome differential diagnosis of a faulty neuromuscular mechanism of swallowing with regurgitation of lipiodol and subsequent glottic aspiration. It also has the advantage of outlining a small fistula which might not be apparent when lipiodol merely trickles down the lumen of the esophagus under no pressure.

TREATMENT.

Before proper operative treatment can be instituted, it is obvious that a diagnosis of fistula must be made, or at least that the presence of a fistula must be very strongly suspected. It is most helpful, however, to be able to localize the fistula. Early diagnosis is important before the inevitable and irreversible changes in the pulmonary system have been produced by repeated attacks of tracheal aspiration. Many of the above methods of diagnosis must be repeated once or possibly twice or even three times before the presence of a congenital tracheoesophageal fistula is satisfactorily eliminated.

When the diagnosis has once been made, ligation and division of the fistula by a direct surgical approach offers the only hope of satisfactory cure. It has been found that chronic or

recurrent pneumonitis is an indication for early operation rather than a contraindication to it. A pulmonary infection will not subside until the communication between the trachea and esophagus has been tightly closed. Gavage feedings should be instituted as soon as the diagnosis is made and employed through as short a period of waiting as possible prior to operation. As soon as the baby's condition permits, thoracotomy should be performed.

In cases of traumatic perforation of the esophagus causing a tracheoesophageal fistula, Clerf and his associates[37,38] have used a bead of silver nitrate or crystals of sodium hydroxide applied by esophagoscopic methods to cauterize the borders of the fistula. These methods, however, do not seem appropriate or feasible in a case of congenital tracheoesophageal fistula in an infant. In such instances direct surgical approach should be used.

The present low operative mortality in cases of tracheoesophageal fistula, even when anastomosis of the two segments of an atresia of the esophagus is carried out, is due in part to improvements in surgical technique. A good deal of credit, however, must be given to a better understanding of the preoperative and postoperative care of the infant. He should be observed in the hospital for at least 24 hours. During this period of time it is important to evaluate his state of nutrition and hydration. If parenteral therapy is indicated, it should be used with understanding. Such infants should not receive unnecessary parenteral fluids. The over-zealous use of saline solution with resultant retention of the sodium ion is a common cause of postoperative edema in these small patients. Five per cent glucose in water, or, if the serum protein is found to be low, concentrated albumin solutions are of value. Complete blood studies are performed preoperatively. In general, if the red blood count is below 4,500,000, a small blood transfusion may be of assistance. If the red blood count is over this more or less arbitrary level, a transfusion of plasma alone might be considered. Each case, however, has to be decided upon its individual findings. Chemotherapy or antibiotic therapy is

instituted prophylactically because of the always present or potential danger of pulmonary infection. Sodium sulfadiazine, penicillin, or aureomycin are given in adequate dosage, depending upon the size of the infant. Atropine is the only other preoperative medication. In cases where cyclopropane anesthesia is used, narcotics are found to be too depressing to the respiratory system of a young infant. When an adequately trained anesthetist is available, cyclopropane has been found to be the most satisfactory anesthetic agent. With this technique a high concentration of oxygen may be given constantly, and with an intratracheal tube in place positive pressure inflation may easily be resorted to if necessary. The advantages of positive pressure are obvious. Cyclopropane may be given either with a tightly fitting face mask or by an intratracheal tube. An intravenous cannula already inserted into an ankle vein before the operation begins is of great value before the operation is completed.

As a result of the pioneer work of Ladd,[5] Lanman[1] and Leven,[16] and the further clinical experience and research of Gross,[39] Haight,[4] Swenson,[41] Ladd,[40] Daniel[42] Humphreys[43] and others, vast improvements in this specialized thoracic surgical technique have been made, and the procedures are now more simplified. Their techniques have been carefully and adequately presented in the literature. Two surgical approaches have been used in performing the thoracotomy; namely, a retropleural or a transpleural exposure. The former method has the advantage of maintaining a sterile, intact pleural cavity so that infection or possible leakage from the site of the fistula does not cause significant trouble, as drainage is to the outside.

In the retropleural approach a curved incision is made on the right between the spinous processes and the scapula. The incision is curved downward and outward. Portions of the third, fourth and fifth ribs are resected posteriorly, but this causes no deformity as the bony defects in an infant are very quickly bridged by callous formation. The mediastinum is opened by reflecting the pleura from the thoracic cage and

the azygos vein is brought into view as it traverses the esophagus. The vein is doubly ligated with black silk and divided between the ligatures. Some difficulty may be encountered in separating the trachea from the esophagus as these two structures are in intimate contact, especially in cases where a fistula is present. Dissection is then carried upward with care, and the vagus nerve must be constantly protected. When the fistula has been identified, it is freed up as completely as possible and doubly ligated with fine black silk. It is then divided between the ligatures and the severed ends of the fistula closed with a row or two of black silk sutures. A simple ligature may result in sloughing and the re-establishment of the patency of the fistula. For this reason a satisfactory closure is recommended in addition to ligation and division. Haight has suggested the repositioning of a bit of fat between the closed surfaces in order to prevent the recanalization of the fistula. He also advises the use of fine suture material and an ample cuff of tissue. A double row of sutures on the cuff of both the trachea and the esophagus is of value if sufficient room is available. Routine closure of the chest wound is employed.

In the transpleural approach a curved incision is made above the right breast and the pectoral muscles transected. The chest is entered in the third interspace by dividing the second and third costal cartilages. The pleura is then opened behind the phrenic nerve and dissection carried backward behind the esophagus. The trachea and esophagus are then carefully separated and the fistula doubly ligated, divided and closed in a manner similar to that used in the retropleural approach. The pleural flap is then sutured with interrupted silk sutures and the chest closed in routine fashion. Just before the skin is completely approximated, all blood and air are aspirated from the chest through a catheter which is quickly withdrawn as the last suture is tied. This helps greatly in the rapid expansion of the lung.

Postoperative care of these little patients is most important. They should be watched very carefully by special nurses

adequately trained in the postoperative care of thoracic surgical patients. Adequate hydration is maintained but the circulation must not be overloaded with fluid. Again the warning regarding over-zealous use of saline solution must be repeated. Frequent determinations of serum protein should be made, and serum albumin or plasma therapy instituted if adequate levels are not maintained. An infant after closure of a tracheoesophageal fistula should be able to take fluids satisfactorily by mouth after recovery from the anesthesia. Chemotherapy and antibiotic therapy are continued to bring the patient through the immediate postoperative period, if possible, without the development of infection.

CASE REPORTS.

Case 1: L. R., Record No. 315641 (courtesy Dr. Swenson). Birthday: March 7, 1947. This three-day-old baby girl was referred to The Children's Hospital on March 10, 1947, with the story that she had appeared perfectly normal at birth except for rapid respirations. On the following day during her first feeding she experienced severe gagging, choking and marked cyanosis. Further feedings precipitated the same difficulties, so oral feedings were stopped, and she was placed on parenteral fluids and intramuscular penicillin therapy. She was then referred to The Children's Hospital for diagnosis and further treatment. At the time of admission the baby was lethargic, but well developed and well nourished. She had a feeble, high-pitched cry. Respirations were rapid and mostly of the abdominal type. The breath sounds were diminished, especially at the left base, and occasional rales were heard throughout both lung fields. The heart sounds were regular and the rhythm was normal. There were no murmurs, and the heart did not appear grossly enlarged. The reflexes were weak, and the baby seemed to be generally hypotonic.

Shortly after admission lipiodol studies showed no obstruction of the esophagus, but halfway between the carina and the inlet of the thorax a definite fistula was visualized running obliquely downward from the trachea into the esophagus (see Figs. 4 and 5). It appeared to be about 1 to 2 mm. in diameter. At the bifurcation of what appeared otherwise to be a normal tracheobronchial tree, the right upper lobe bronchus originated from the trachea (see Fig. 6). Fluoroscopic examination of the chest showed a heart which was considerably enlarged and produced a slight posterior displacement of the esophagus. The lungs were irregularly hyperventilated with moderate engorgement of vessels, but no definite aspiration pneumonia could be detected. It was felt that the baby had a congenital malformation of the heart with a probable shunt to the pulmonary circulation, possibly on the basis of a patent ductus arteriosus or an interauricular septal defect. On the following day under cyclopropane anesthesia, thoracotomy was performed using the retropleural approach. The esophagus appeared grossly normal. The opening of the fistula into the trachea seemed to be about 3 to 5 mm., but was smaller on the esophageal side. It was ligated, divided, and the tracheal and esophageal openings closed. The baby seemed to withstand the proce-

dure satisfactorily. On the following day, however, she again developed cyanosis with feedings, and it was felt advisable to perform a temporary gastrostomy. The attacks of cyanosis continued, however, in spite of only gastrostomy feedings. Serum protein was only 4.6 mgm. per cent, so serum albumin was given in the hope of reducing the generalized edema which had developed. She was kept on penicillin and streptomycin therapy and in oxygen most of the time. The pneumonia became very widespread, and seemed especially resistant to treatment. The baby's course was downhill. She died on the fifth day after closure of the tracheoesophageal fistula.

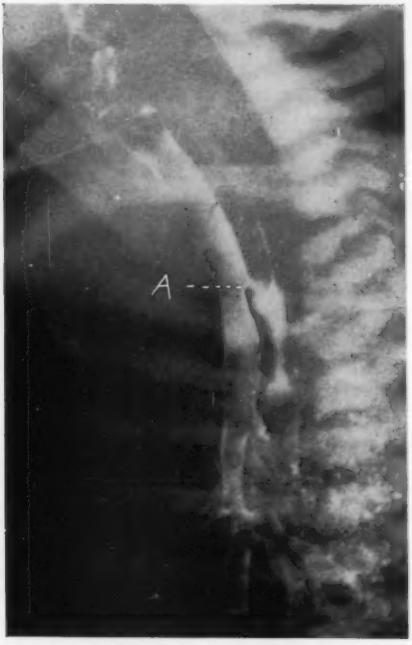

Fig. 4. Lateral Roentgenogram of intratracheal injection of lipiodol demonstrates tracheoesophageal fistula (A).

Postmortem examination showed a very extensive bilateral interstitial type of bronchopneumonia, as well as evidence of aspiration pneumonia (see Figs. 7 and 8). There was also marked pulmonary edema. The right upper lobe bronchus was found to originate from the trachea as had been noted in the Roentgenological findings. The heart was grossly enlarged due to hypertrophy of its muscle, and there was a patent ductus arteriosus. An incidental finding was a Meckel's diverticulum. The esophagus was normal, and the fistula had apparently been satisfactorily closed,

with no evidence of mediastinal infection (see Fig. 9—shows the histological aspect of the repair). The immediate cause of death in this case was felt to be the widespread bronchopneumonia which involved all lobes of both lungs (see Figs. 10 and 11—demonstrate the organs of the neck and chest removed at the time of the postmortem examination).

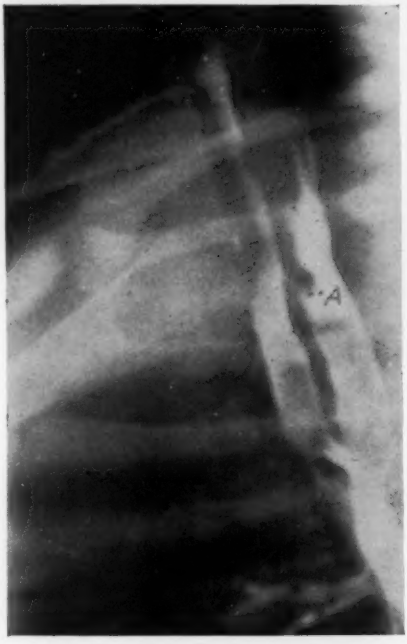

Fig. 5. Close-up view of Fig. 4.

Case 2: E. I., Record No. 323388 (courtesy Dr. Swenson). Birthday: Sept. 17, 1947. This five-day-old baby boy was referred to The Children's Hospital on Sept. 22, 1947, because of attacks of vomiting and cyanosis since birth. Several hours after birth it was noted that he had an excessive amount of frothy saliva. He was started on a formula which he promptly vomited and during the procedure became very cyanotic. Vomiting and cyanosis were associated with further feedings, so hydration was maintained by parenteral fluids only. He was referred to the hospital for diagnosis and treatment of probable esophageal obstruction. At the time of admission the baby appeared well developed and well nourished and in a fairly good state of hydration. He manifested a slightly wheezing type of respiration, but the rate was normal, and there was no evidence of retraction of the chest or cyanosis. He had an increased amount of mucus in his mouth and pharynx, and numerous coarse rhonchi in both lungs.

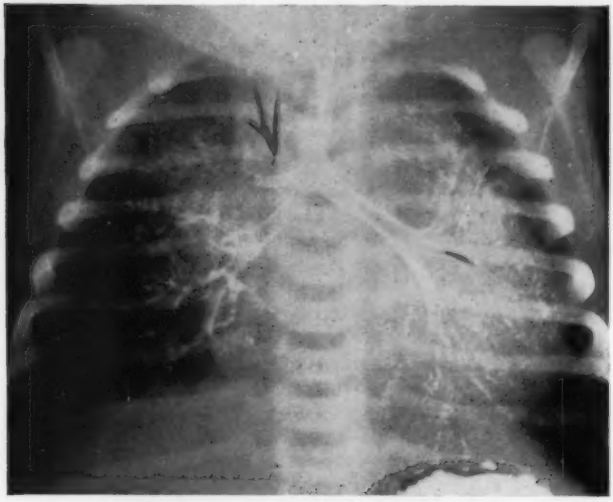

Fig. 6. A-P Roentgenogram of intratracheal injection of lipiodol showing right upper lobe bronchus arising from trachea.

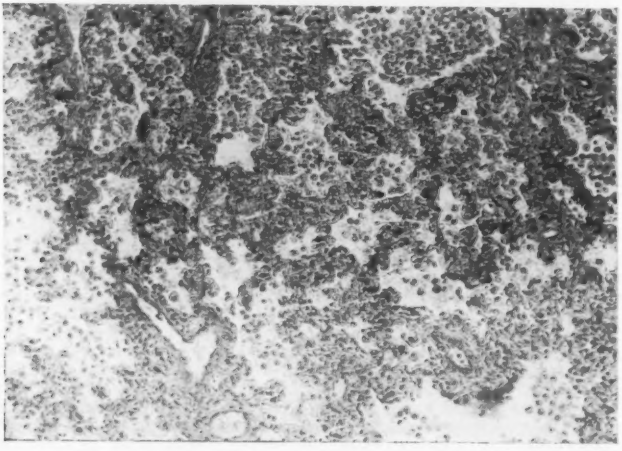

Fig. 7. Photomicrograph (×160) of lung showing extensive bronchopneumonia with numerous macrophages in the alveolar spaces.

Shortly after admission the patient was given a swallow of lipiodol which showed an esophagus considerably narrowed at the fourth thoracic vertebra, but the lipiodol passed through this area into the stomach. There was a large amount of gas distributed throughout the intestinal tract. The lungs showed considerable peribronchial pneumonic infiltration throughout the entire chest, more marked in the right upper lobe, and probably secondary to aspiration. Some lipiodol was noted in the trachea and bronchi, but a fistula could not be demonstrated. The baby was kept on parenteral feedings and penicillin therapy. Because of excessive mucus which required frequent aspiration, coupled with several chok-

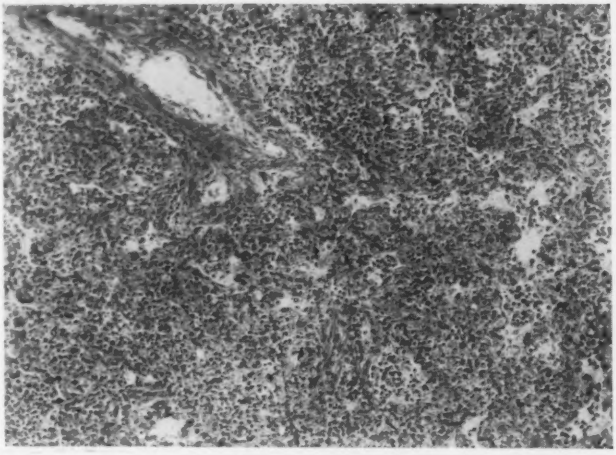

Fig. 8. Photomicrograph of lung (×120) showing bronchopneumonia, with marked edema and alveolar exudate.

ing and gagging spells and a troublesome cough, it was felt advisable to perform a gastrostomy. This was done on the day following admission. An interesting finding is the fact that the stomach seemed to be so distended from time to time that it was necessary to deflate it by periodically opening the gastrostomy tube. Roentgenologic evidence of pneumonitis persisted although the baby's condition remained satisfactory. On the thirteenth day after the gastrostomy the esophagus was dilated by retrograde Tucker bougies, but it was possible to go through only a No. 10 Fr. Penicillin and streptomycin were given regularly in full dosage and oxygen and humidification were occasionally administered. Eleven days later his general condition as well as the Roentgenologic picture had improved, and it was again possible to dilate the esophagus, this time going through a No. 22 Fr. bougie. After the dilatation, further attempts were made to feed the patient liquids by mouth, but he still continued to have attacks of choking and cyanosis. It was generally supposed that he had a tracheoesophageal fistula which had not been demonstrated. The trachea and bronchi were then outlined by an intra-

tracheal injection of lipiodol. No fistulous tract was demonstrated, but it was noted that "the lipiodol seemed to spill over into the esophagus." Two days later on a third attempt a narrow tracheoesophageal fistula was successfully demonstrated (see Figs. 12 and 13). It was 5 mm. in length, possibly 1 or 2 mm. in diameter, and ran from the posterior aspect of the trachea downward and backward, where it joined the esophagus at

Fig. 9. Photomicrograph (×40) showing satisfactory healing at site of fistula repair.

the level of the second thoracic vertebra. Two days later under intratracheal cyclopropane anesthesia a thoracotomy was performed using a retropleural approach. The esophagus appeared grossly normal except for slight diminution in diameter in the region of the bifurcation of the trachea. It was tightly adherent to the trachea at this point, but it was possible to separate it satisfactorily. The fistulous tract was demonstrated as outlined by the lipiodol, and was ligated, divided and the tracheal and esophageal openings closed with two rows of black silk sutures.

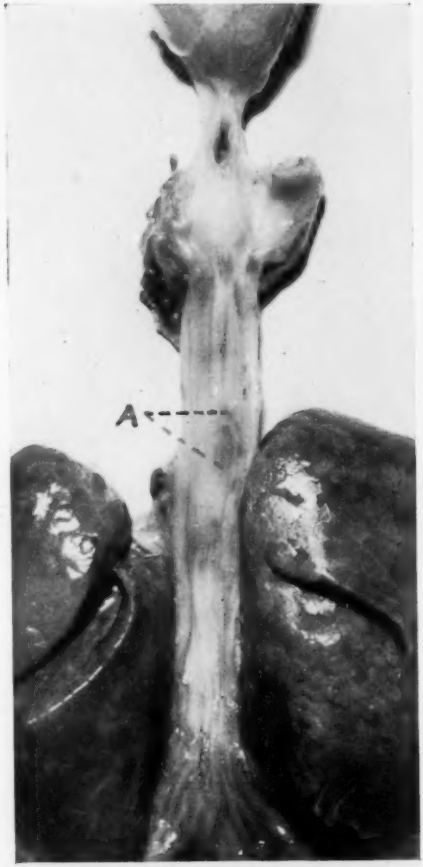

Fig. 10. Posterior view of autopsy specimen showing site of tracheoesophageal fistula repair (A) from esophageal side. Note edema of lungs.

The patient withstood the procedure satisfactorily and was discharged on the eighth day after the operation. Following the closure of the fistula he had no further trouble with excessive saliva and no regurgitation of feedings. The gastrostomy was closed three months later. He continued to gain weight and swallow normally. When he was last seen on July 11, 1950, at the age of two years and 10 months, there were no complaints, and he was able to take a perfectly normal solid diet without difficulty.

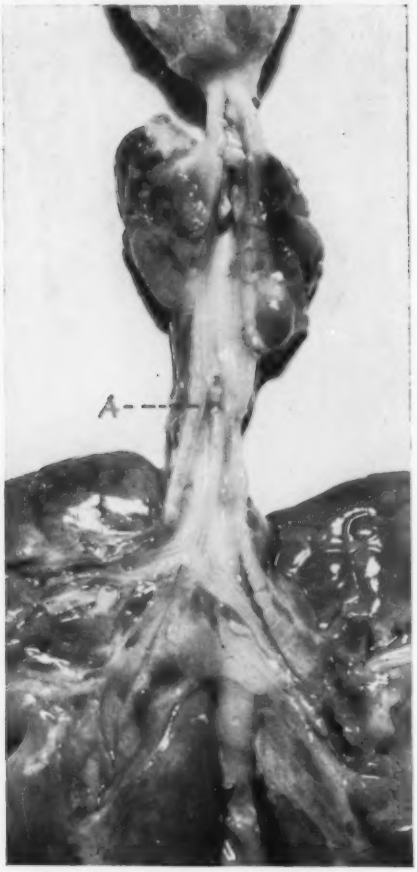

Fig. 11. Anterior view of fistula repair (A) from tracheal side.

Case 3: C. C., Record No. 327588 (courtesy Dr. Gross). Birthday: Dec. 16, 1947. This one-month-old baby girl was referred to The Children's Hospital on Jan. 17, 1948, for difficulty in swallowing and attacks of cyanosis. She had been apparently normal until the third day of life, at which time she choked during her first feeding. Attacks of choking and cyanosis occurred with subsequent feeding. On the fourteenth day of life a lipiodol swallow showed a filling defect in the posterior portion of the esophagus consistent with an aberrant subclavian artery. She was referred to The Children's Hospital and made the rather long trip by plane.

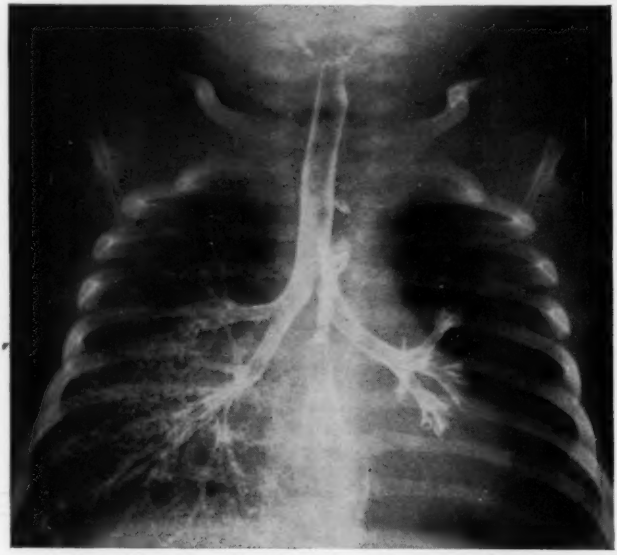

Fig. 12. A-P Roentgenogram of intratracheal injection of lipiodol outlining esophagus as well.

During the flight she had one episode of cyanosis, for which oxygen and metrazol were used. The only other medication had been penicillin for the three days following the severest choking episode at two weeks of age. At the time of admission the baby was well developed, well nourished and apparently satisfactorily hydrated. Her respirations were somewhat rapid, but the respiratory excursions were normal and the lungs were clear on auscultation and percussion. The heart appeared normal on clinical examination.

Shortly after admission a lipiodol swallow revealed a posterior esophageal defect running obliquely upward from left to right at the level of the third thoracic vertebra. This was interpreted as the characteristic appearance of an aberrant right subclavian artery. There was only slight delay in passage of lipiodol through the esophagus proximal to the

defect and no evidence of obstruction. During forced expiration there was a flow of lipiodol into the trachea at the second thoracic vertebra through a minute fistula measuring at most 1 to 1.5 mm. in diameter (see Fig. 14). The trachea itself presented no displacement or compression. The lung picture was characteristic of aspiration pneumonia of moderate severity.

Because of the pneumonitis, after the patient had been on sulfadiazine and penicillin for two days, a gastrostomy was performed under cyclopropane anesthesia. The pneumonitis improved remarkably after this procedure. Forty-eight hours later, thoracotomy was performed under

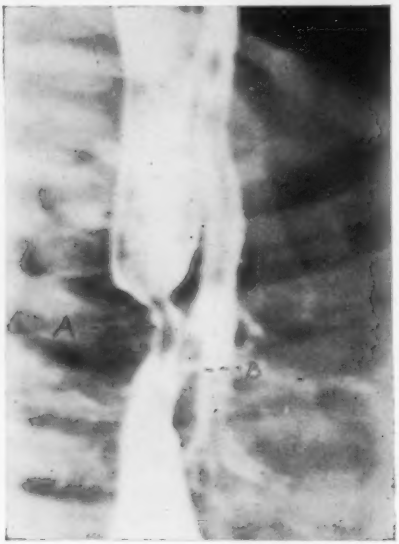

Fig. 13. Lateral Roentgenogram of same case. Note marked stenosis of esophagus (A) and fistula (B).

cyclopropane anesthesia given by an intratracheal tube. A transthoracic approach was used. The aberrant vessel was readily identified, doubly ligated and divided. The trachea and esophagus were carefully dissected and the fistula also readily identified and ligated. It was then divided, and the esophageal and tracheal openings closed. Blood and air were aspirated from the thoracic cavity through a catheter which was withdrawn just before the skin was tightly closed.

The infant had a satisfactory postoperative course except for a brief flare-up of the pneumonia in spite of continuing chemotherapy. Nutrition and adequate serum protein levels were maintained on gastrostomy feedings coupled with several blood transfusions. On the thirteenth postoperative day repeat lipiodol studies of the esophagus showed only slight

persistence of the indentation in the posterior wall at the level of the divided subclavian artery. The notch was smaller and more shallow than on previous examination. The fistulous tract could not be identified. At the time of discharge from the hospital on the thirteenth postoperative day the baby was able to take many of her feedings by mouth. On the twenty-eighth postoperative day it was possible to remove the gastrostomy tube since the baby was swallowing perfectly normally. A two-year follow-up report was obtained in March, 1950. Her condition at that time was excellent, and she was taking a normal diet without difficulties.

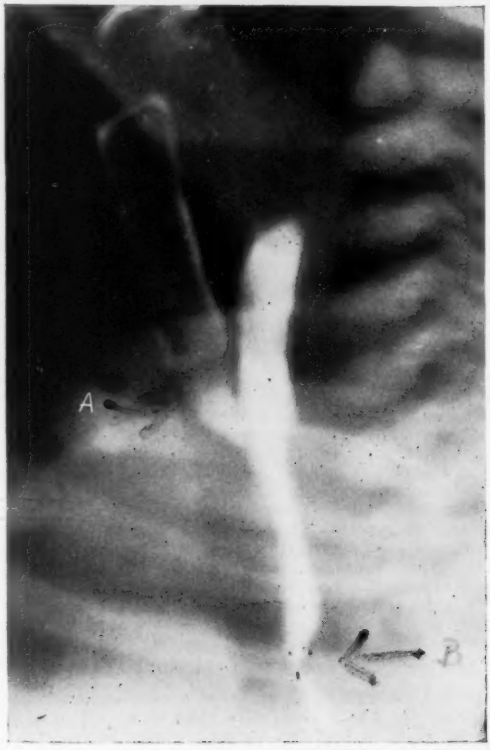

Fig. 14. Lipiodol Roentgenogram showing tracheoesophageal fistula (A) and posterior filling defect of esophagus produced by aberrant right subclavian artery (B).

Case 4: L. S. D., Record No. 224653. Birthday: Feb. 18, 1949. This 10-month-old baby boy was first seen in the out-patient department of The Children's Hospital on Dec. 17, 1949, with the story that since birth he

had breathed heavily and noisily and seemed to have a constant upper respiratory infection. During the first month of life he had been kept in oxygen a good deal of the time. At three months of age he developed pneumonia and spent another month in a hospital. He had had no swallowing difficulties at any time. Whenever he started to gain weight, however, he developed another respiratory infection which resulted in further weight loss. He was slow in development and at the age of 10 months had no teeth and was unable to sit up alone. At the time of admission to the medical out-patient department of The Children's Hospital the baby was 10 months of age, but weighed only 12 pounds, six ounces. Height was 64.6 cm., and both weight and height were below the lowest percentile rating. His eyes had a definite Mongoloid appearance, and the fontanels were still open. The tongue was very large. The heart appeared somewhat enlarged, and the apex impulse was 1 to 2 cm. to the left of the midclavian line. The rate was 140 and there was marked sinus arrhythmia. There was a Grade II pulmonary systolic murmur, but no diastolic murmur was heard. The extremities showed marked general hypotonicity, the hands were wide and thick, and large spaces were noted between the first and second toes on both feet. The reflexes were equal and normally active. It was felt that the baby was Mongoloid and that he also had congenital heart disease. Fluoroscopic examination of the chest showed a right middle lobe pneumonia. There was right ventricular hypertrophy and the auricles were enlarged. The heart seemed to be increased in its transverse diameter and showed an unusual configuration with marked elevation of the apex. It was felt that the patient had an aberrant right subclavian artery and a left to right shunt through a patent interauricular septum. The patient was followed by his local doctor until the pneumonia had subsided and then was admitted to the hospital on Feb. 1, 1950, for more complete study. Because of the cardiac and vascular situation the baby was given a lipiodol swallow in order to determine whether there was any compression of the esophagus. A typical oblique filling defect was noted on the posterior aspect of the esophagus running upward from left to right at the level of the aortic arch (see Fig. 15). In addition lipiodol was seen to pass from the esophagus into the trachea through a definite fistulous communication on the anterior aspect of the esophagus just below the jugular notch. There was no esophageal obstruction, and in addition to vascular engorgement considerable irregularity of aeration of the lungs with atelectasis of the right upper lobe was noted. The heart was grossly enlarged both to the right and left and was abnormal in contour. There was a right ventricular hypertrophy with enlargement of both auricles and considerable fullness in the pulmonary artery area with engorgement of the intrapulmonary vessels. The appearance was that of an aberrant right subclavian artery arising as the last branch of the aortic arch and passing to the right behind the esophagus. It was felt that the patient in addition had a large interauricular septal defect.

Because of the findings of such a severe cardiac abnormality and the fact that the patient was a Mongolian idiot as well, it was decided that he was a poor surgical risk for operation upon either the constricting artery or the tracheoesophageal fistula. For this reason he was discharged to the care of his local doctor. On attempted follow-up examination a few weeks later it was discovered that the baby had died at home. No autopsy had been performed.

This baby represented a combination of several congenital anomalies. He had a severe malformation of the heart with an anomalous vascular system producing a probable right subclavian artery which because of its

position caused compression of the esophagus. The baby from all physical appearances was a Mongolian idiot and in addition had a congenital tracheoesophageal fistula without an associated atresia of the esophagus. Unfortunately, however, it was not possible to obtain positive confirmation of this latter finding either by surgery or by postmortem examination.

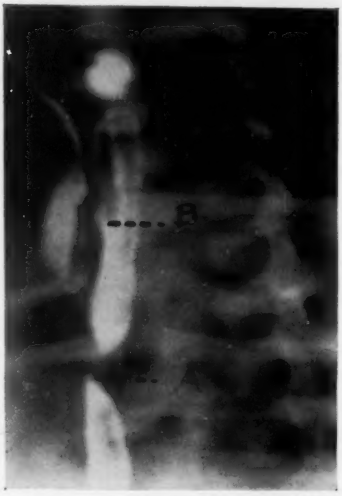

Fig. 15. Lipiodol study of esophagus showing posterior filling defect produced by aberrant right subclavian artery (A), and tracheoesophageal fistula (B).

Case 5: C. F., Record No. A-46-23 (courtesy Dr. Farber). This seven and one-half months old baby girl was brought to The Children's Hospital on Feb. 6, 1946, dead on arrival. The findings of the postmortem examination are presented in some detail as this was the first case of tracheoesophageal fistula with a normal continuity of the esophagus seen at this hospital. The baby had been admitted to another hospital two days before death with a complaint of labored breathing. Six weeks previously she had been placed in a foster home and physical examination at that time was said to have been normal. Following placement the patient had been unable to swallow anything but liquids. There had been associated respiratory difficulty and a spasmodic nonproductive cough. Unfortunately further details of the history were not available. On the day of admission to the other hospital the baby was said to have been dehydrated and in acute respiratory distress. Roentgenograms showed a metallic foreign body interpreted as probably in the left main bronchus or possibly in the esophagus. Endoscopy was performed and during the procedure a foreign body was said to have been "dislodged but not removed." Transfer to The Children's Hospital was advised, but the patient died in the ambulance en route to the latter institution. The postmortem examination findings are as follows:

The body was that of a well developed and fairly well nourished female baby 61 cm. long (normal for this age is 65 cm.). No mediastinal emphysema or edema was noted. The lungs showed very extensive bronchopneumonia with little remaining aerated parenchyma. B. Friedlander, Type B, was recovered in pure culture, and the presence of gas bubbles in the blood stream was interpreted as being related to a probable B. Friedlander's bacteremia. Cultures from the blood stream, however, did not grow out any organisms. In the opinion of the pathologist there was no reason to believe that an air embolus had occurred before death. There was considerable patchy congestion of the mucosal surfaces of the lungs and large amounts of purulent material in the bronchi and bronchioles. On microscopic examination there was very little evidence of general aspiration pneumonitis, although in local areas the picture was rather typical. The diagnosis was that of a marked chronic purulent bronchitis. Acute inflammation and edema of the tracheobronchial lymph nodes were noted. Except for the local examination of the trachea and the esophagus, the only other congenital anomalies were double ureters and pelves of the kidneys on both sides, and a patent foramen ovale.

Six and five-tenth cm. below the proximal extremity and 6.8 cm. above the distal extremity of the esophagus was a fistula 0.4 cm. in diameter. The fistula was round and its periphery was smooth and regular (see Fig. 16A) . There was no gross evidence of inflammatory changes. It opened directly into the trachea just above the carina. In the region 0.7 cm. to the right and inferior to the fistula was an irregular, round defect in the esophageal wall 0.4 cm. in diameter. Just below the fistula was noted a small redundant flap of mucosa (see Fig. 17A) about 0.5 cm. in height and 0.5 cm. in diameter. The mucosa immediately adjacent to it was torn, eroded and necrotic. Lower down in the esophagus were several smaller longitudinal erosions (see Fig. 17B). On splitting the trachea anteriorly, the fistula was seen 1.3 cm. above the carina, measuring 0.4 cm. in diameter. At the carina and along the right borders of the trachea and esophagus were noted chains of lymph nodes which were dark brownish-red in color, moderately firm, and varied from 0.4 to 1.2 cm.

On microscopic examination of the irregular, ragged defect in the esophageal wall there was evidence of edema, acute and chronic inflammation, and necrosis. Numerous dilated capillaries were noted growing toward the necrotic layer and these vessels showed hyperplastic endothelial lining. There was a rather long segment of mucosa including part of the muscularis mucosae torn away from the underlying layers and forming an elongated tab in the region adjacent to the erosion. The exposed surfaces were lined by a layer of fibrinoid necrosis in which were numerous colonies of bacilli.

Examination of the margin of the fistula into the trachea showed that the connective tissue layers of both the trachea and esophagus were continuous. The stratified squamous epithelium of the esophagus extended for some distance into the trachea where it merged into a thick, ciliated, pseudostratified columnar type of epithelium. There was slight edema associated with infiltration by lymphocytes and plasma cells, but no evidence of necrosis or an acute inflammatory cell response. This patient demonstrated a congenital tracheoesophageal fistula without atresia of the esophagus, and the perforation on the posterior wall of the esophagus below the fistula appeared to be traumatic and quite recent. It was considered that the perforation was the result of the fact that a foreign body had remained in the esophagus for some time and had eroded its wall, or that it was the result of the difficult endoscopic proce-

dures at the other hospital. The foreign body must have been dislodged and passed through the intestinal tract prior to death. At any rate, it was not discovered by careful search of the entire body at the time of the autopsy.

Fig. 16. Autopsy specimen of neck and thoracic organs opened posteriorly showing mucosal surface of esophagus. Note fistulous opening (A) and yellow bronchopneumonic mottling (B) most striking on posterior surface of right lung.

This patient represented the first case of congenital tracheoesophageal fistula with a normal continuity of the esophagus seen in the pathology department of The Children's Hospital. It is interesting that until that date (1946) nearly 100 cases of congenital tracheoesophageal fistula had gone through this department. None of them, however, had shown an

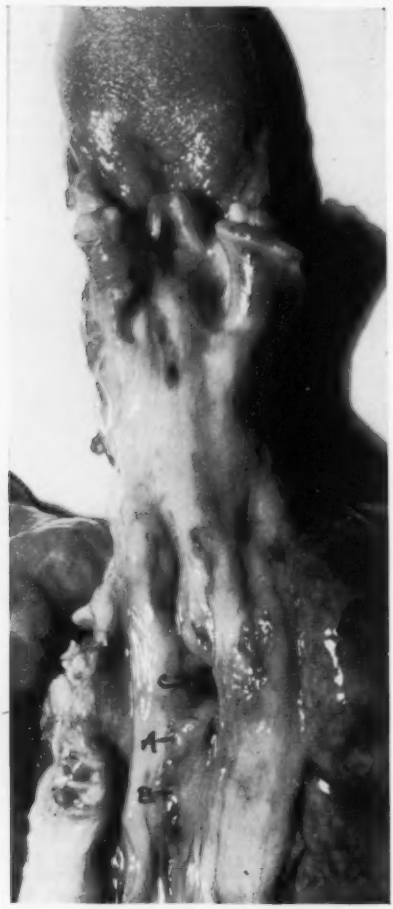

Fig. 17. Close-up of fistula (C). Below fistula is an elevated tab of mucosa (A); and an irregular longitudinal erosion (B).

otherwise normal esophagus. Since 1946 four other cases reported in this series were diagnosed clinically, and three of them were treated surgically.

SUMMARY AND CONCLUSIONS.

1. Congenital tracheoesophageal fistula without the usually associated atresia of the esophagus is not so rare as the literature indicates. Only two cases have so far been reported in which the diagnosis was made and a successful operation performed. In neither of these was diagnosis made during infancy.

2. The embryology and possible causative factors are discussed.

   a. The most plausible theory is that there is an arrest in the development of the lateral cell masses which separate the primitive foregut tube into the esophagus dorsally and the trachea ventrally.

   b. A genetic cause of this arrest cannot be eliminated, and environmental factors should also be considered.

   c. The fistula is probably well established by the seventh to eighth week of fetal life (6 to 8 mm. stage) as embryological studies have demonstrated.

3. A brief description of the gross and microscopic pathology is included.

4. The symptomatology is not constant. Diagnosis should be suspected in an infant who has frequent attacks of choking, with or without cyanosis during feedings.

5. Diagnosis is often very difficult, and repetition of many of the diagnostic procedures is necessary. Methods employed consist of the passage of catheters, lipiodol studies of the trachea, and esophagus by film and fluoroscope, and repeated endoscopic procedures, possibly with dyes or with lipiodol under pressure. The passage of a catheter does not eliminate the possibility of a tracheoesophageal fistula.

6. The procedure of surgical ligation, division and closure of the fistula is the only recommended method of treatment, and should be carried out as soon as the diagnosis is made and the patient's condition is satisfactory. Preoperative and postoperative care is discussed.

7. Five cases of congenital tracheoesophageal fistula without atresia of the esophagus are included. In all five instances the diagnosis was made during the first year of life, and in three the fistula was closed by surgery before the end of the second month. Two patients are living and well, and one died postoperatively. The findings of the postmortem examination are presented. The fourth patient was not operated upon because of a severe congenital cardiac condition, and died at home. The fifth is an autopsy report of an infant brought to the hospital, dead on admission. Lipiodol Roentgenograms and postmortem photographs have been included in the presentation.

BIBLIOGRAPHY.

1. Lanman, T. H.: Congenital Atresia of the Esophagus: A Study of 32 Cases. *Arch. Surg.*, 41:1060, 1940.

2. Swenson, O.: Diagnosis and Treatment of Atresia of the Esophagus and Tracheoesophageal Fistula. *Pediat.*, 1:195, 1948.

3. Imperatori, C. J.: Congenital Tracheoesophageal Fistula Without Atresia of the Esophagus. Report of a Case with Plastic Closure and Cure. *Arch. Otolaryngol.*, 30:352, 1939.

4. Haight, C.: Congenital Tracheoesophageal Fistula Without Esophageal Atresia. *Jour. Thoracic Surg.*, 17:600, 1948.

5. Ladd, W. E.: The Surgical Treatment of Esophageal Atresia and Tracheoesophageal Fistula. *New Eng. Jour. Med.*, 230:625, 1944.

6. Mackenzie, M.: A Manual of Diseases of the Throat and Nose. New York, N. Y.: Wm. Wood, 1884.

7. Gibson, T.: Anatomy of Human Bodies Epitomized. London, 1703.

8. Vogt, E. C.: Congenital Esophageal Atresia. *Amer. Jour. Roent.*, 22:463, 1929.

9. Lamb, D. S.: A Fatal Case of Congenital Tracheoesophageal Fistula. *Phila. Med. Times*, 3:705, 1873.

10. Cautley, E.: Malformations of the Esophagus. *Brit. Jour. Child. Dis.*, 14:1, 1917.

11. Plass, E. D.: Congenital Atresia of the Esophagus and Tracheoesophageal Fistula. *Johns Hopkins Hosp. Rep.*, 18:259, 1919.

12. Holt, J. F.; Haight, C., and Hodges, F. J.: Congenital Atresia of the Esophagus and Tracheoesophageal Fistula. *Radiol.*, 47:457, 1946.

13. Negus, V. E.: Esophagus from a Middle-Aged Man Showing a Congenital Opening into the Trachea. *Jour. Laryngol. and Otol.*, 44:184, 1929.

14. Rosenthal, A. H.: Congenital Atresia of the Esophagus with Tracheoesophageal Fistula: Report of Eight Cases. *Arch. Path.*, 12:756, 1931.

15. McKinney, R.: Tracheoesophageal Fistula, Probably Congenital, in a Woman 58 Years of Age. *Ann. Otol., Rhinol. and Laryngol.*, 42:1237, 1933.

16. Leven, N. L.: Congenital Atresia of the Esophagus with Tracheoesophageal Fistula. Report of Successful Extrapleural Ligation of Fistulous Communication and Cervical Esophagostomy. *Jour. Thoracic Surg.*, 10:648, 1941.

17. Gengenbach, F. P., and Dobos, E. I.: Congenital Tracheoesophageal Fistula. *Jour. Pediat.*, 19:644, 1941.

18. Leven, N. L., and Lannin, B. G.: Congenital Atresia and Tracheoesophageal Fistula. *Jour. Lancet*, 65:179, 1945.

19. Ingalls, T. H., and Prindle, R. A.: Esophageal Atresia with Tracheoesophageal Fistula; Epidemiologic and Teratologic Implications. *New Eng. Jour. Med.*, 240:987, 1949.

20. Grieve, J. G., and McDermott, J. G.: Congenital Atresia of the Esophagus in Two Brothers. *Can. Med. Assn. Jour.*, 41:185, 1939.

21. Ballantyne, J. W.: Manual of Antenatal Pathology and Hygiene: The Embryo. Edinburgh: W. Green & Sons, 1904.

22. Ysander, F.: Human Diploterata. *Upsala Lakaref. Forh.*, 30:195, 319, 1924-1925.

23. Gruenwald, P.: Case of Atresia of Esophagus Combined with Tracheoesophageal Fistula in 9 Mm. Human Embryo and Its Embryological Explanation. *Anat. Rec.*, 78:293, 1940.

24. Minot, C. S.: A Laboratory Textbook of Embryology. Philadelphia, Pa.: Blakiston, 1910.

25. Kreuter, E.: Die angeborenen Verschliebungen und Verengerungen des Darmkanals im Lichte der Entwicklungsgeschichte. *Deut. Ztsch. f. Chirurg.*, 79:1, 1905.

26. Forssner, H.: Die angeborenen Darm- und Oesophagus-atresien. *Anat. Hefte*, 34, 1907 (quoted by Schmitz, J. A. Virchow's *Arch. f. Path. Anat.*, 247-278, 1923).

27. Schridde, H.: Ueber Epithelproliferationen in der Menschlichen Speiserohre. Virchow's *Arch. f. Path. Anat.*, 191:178, 1908.

28. Keith, A., and Spicer, J. E.: Three Cases of Malformation of the Tracheoesophageal Septum. *Jour. Anat. and Physiol.*, 41:52, 1906-1907.

29. Ingalls, T. H., and Gordon, J. E.: Epidemiologic Implications of Developmental Arrests. *Amer. Jour. Med. Sci.*, 214:322, 1947.

30. Brigham, R. O.: Atresia of the Esophagus. *Amer. Med.*, 35:297, 1929.

31. Meyer, H.: Esophagusatresie und Hydramnion. *Zentralbl. f. Gynak.*, 53:1562, 1929.

32. Scheurer, D.: Ein Fall von Kongenitaler Oesophagusatresie mit Oesophago-Tracheal-fistel. *Schweiz. Md. Wchnschr.*, 9:1079, 1928.

33. Strong, R. A., and Cummins, H.: Congenital Atresia of the Esophagus with Tracheoesophageal Fistula. *Amer. Jour. Dis. Child.*, 47:1299, 1934.

34. Reinhoff, F.: Congenital Arteriovenous Fistula. *Bull. Johns Hopkins Hosp.*, 25:271, 1924.

35. Rector, L. E., and Connerley, M. L.: Aberrant Mucosa in Esophagus in Infants and Children. *Arch. Path.*, 31:285, 1941.

36. Abbott, O. A.: Abnormal Esophageal Communications: Their Types, Diagnosis and Therapy. *Jour. Thoracic Surg.*, 14:382, 1945.

37. Clerf, L. H.: Bronchoesophageal Fistula. Report of a Case. *Ann. Otol., Rhinol. and Laryngol.*, 42:920, 1933.

38. Clerf, L. H.; Cooley, E. E., and O'Keefe, J. J.: Esophagobronchial Fistula. *Surg., Gynec. and Obst.*, 77:615, 1943.

39. Gross, R. E., and Scott, H. W.: Correction of Esophageal Atresia and Tracheoesophageal Fistula by Closure of Fistula and Oblique Anastomosis of Esophageal Segments. *Surg., Gynec. and Obst.*, 82:518, 1946.

40. Swenson, O., and Ladd, W. E.: Esophageal Atresia and Tracheoesophageal Fistula. *Ann. Surg.*, 125:23, 1947.

41. Swenson, O.: End-to-End Anastomosis of the Esophagus for Esophageal Atresia. *Surg.*, 22:324, 1947.

42. Daniel, R. A.: Congenital Atresia of the Esophagus with Tracheoesophageal Fistula. *Ann., Surg.*, 120:764, 1944.

43. Humphrey, G. H. :II—Surgical Treatment of Congenital Atresia of the Esophagus. *Surg.*, 15:801, 1944.

THE CLINICAL AND EXPERIMENTAL COMPARISON OF COCAINE AND PONTOCAINE AS TOPICAL ANESTHETICS IN OTOLARYNGOLOGICAL PRACTICE.*

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The purpose of this presentation is to discuss some clinical and experimental findings on the relative toxicity and potency of these two drugs which are so commonly used for topical anesthesia in the practice of otolaryngology. A survey was conducted in 1928 by the American Medical Association to compare the local anesthetics in general use at that time. Since then we have had the introduction of tetracaine, commonly known as pontocaine. A comparison of pontocaine with cocaine is all that will be discussed.

The problem was first approached as an experimental investigation by Dr. Eric Stark in the Dapartment of Pharmacology of the University of Toronto, under Prof. Ferguson. The clinical experiments were conducted in the Department of Otolaryngology, Toronto General Hospital. A survey was conducted by means of a questionnaire sent to the larger clinics in the United States and Canada.

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EXPERIMENTAL STUDIES.

The details of the experimental studies will not be given in this paper, but only a summary of the conclusions made from their analysis. The two drugs considered were used in their pure forms and in various strengths. They were also tested with their added preservatives as sold in the drug market. Local application to the larynx and carina by means of a specially designed laryngoscope and bronchoscope was used, and direct application by drop instillation after external isolation of the trachea. The experimental animals used were dogs, guinea pigs and rabbits, and many results were discarded because of factors which were considered to be due to extraneous causes. A summary only is given under the following headings: *1.* Measurements of toxicity. *2.* Fatal doses. *3.* Effects on mucous membrane. *4.* Estimation of the potency on human mucosa.

1. Measurements of Toxicity.

Reports on the toxicity of cocaine and pontocaine when injected into animals give figures for the median lethal dose of pontocaine which vary from three-fourths to one-third of those for cocaine.[1-3] In other words, the toxicity of pontocaine is given as one and three-tenths to three times that of cocaine. If these figures are applicable to humans, one would expect pontocaine to be a safer drug clinically than cocaine, since pontocaine is customarily applied in 1 to 2 per cent solution, or about one-fifth the concentration at which cocaine is ordinarily used.

We felt, however, that figures for lethal doses by injection were hardly fair for estimating relative toxicity of drugs used topically; consequently, an experiment was designed to measure the lethal doses of these drugs by application to the tracheal mucosa of the guinea pig.

Large guinea pigs, weighing 0.7 to 1.3 kgm., were secured in a supine position. The trachea was exposed through a small incision made after infiltration of the skin with a small

amount of 0.5 per cent procaine hydrochloride. Cocaine as a 10 per cent solution of the hydrochloride, and pontocaine as 2 per cent solution of the hydrochloride, were injected into the trachea, drop by drop, through a 26 gauge needle on a tuberculin syringe. The rate of administration was timed to cause death with typical convulsions and respiratory failure in 10 to 15 minutes. The total volume injected seldom exceeded 0.6 cc., *i.e.*, not enough to drown the animals.

2. Fatal Doses.

The mean fatal dose for cocaine HCl (11 animals) was 24 mg./kg., with a standard deviation of 4.6 mg./kg. That of pontocaine HCl (11 animals) was 7.3 mg./kg. with a standard deviation of 0.2 mg. The toxicity of pontocaine HCl was thus found to be about three and four-tenths times that of cocaine HCl.

3. Effects on Mucosa.

In preliminary experiments with pontocaine injected into the trachea very slowly, the animals often died from respiratory obstruction. This proved to be due to swelling and desquamation of the tracheal mucosa. This unexpected result was obtained using many different solutions of pontocaine ranging in strength from 0.5 to 2 per cent, both freshly made and as supplied ready-made by the manufacturers. A similar desquamating action was observed on tracheal mucosa of rabbits. Although this peculiar irritating or histotoxic action has not been observed on human tracheal mucosa, it may be related to the clinical observation that pontocaine has a tendency to provoke asthmatic attacks in persons subject to this affliction.

In summary, these measurements of fatal doses support earlier ones in suggesting that pontocaine should have a greater margin of safety than cocaine when used at the customary concentrations. The reaction of the tracheal mucous membrane served a warning, however, that the acute lethal doses might not tell the whole story of potential toxicity.

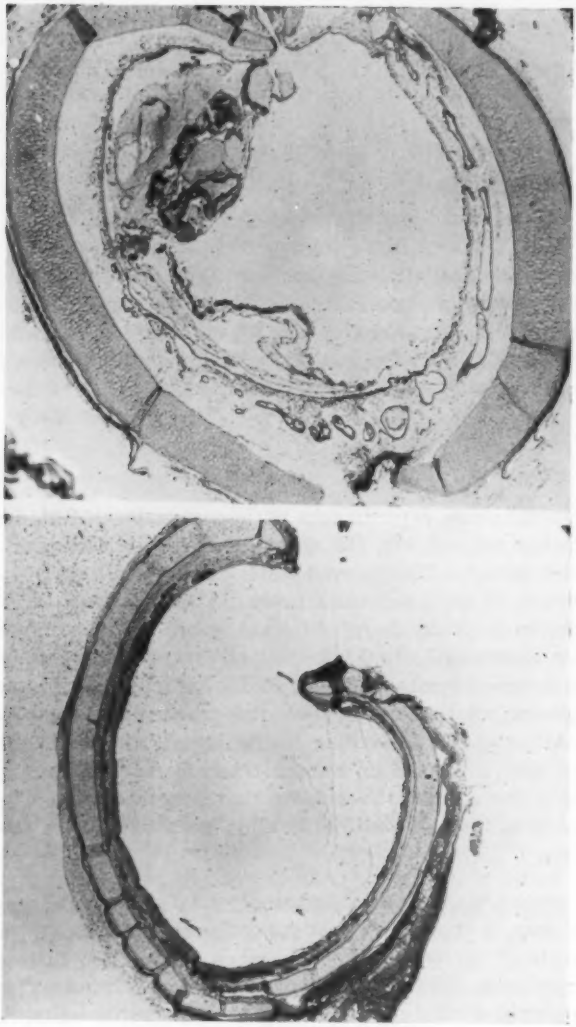

FIG. 1. Guinea pig trachea (low power). Section on left shows normal cross-section. Section on right shows edema and marked narrowing of lumen after pontocaine instillation.

4. Estimation of Potency on Human Mucosa.

Cocaine HCl 5 per cent was compared with pontocaine HCl 1 per cent by application to the nasal septa of patients. Pieces of blotting paper, half an inch square, were moistened with four drops of the solution under test and applied to healthy mucous membrane one-half to three-fourths of an inch behind

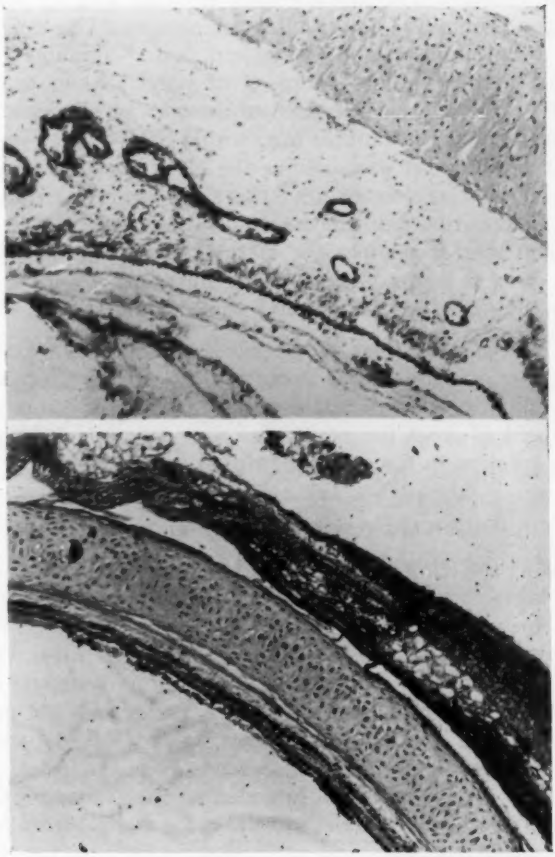

Fig. 2. Guinea pig trachea (high power). Section on left shows narrow submucosa and normal ciliated epithelium. Section on right was after the instillation of pontocaine. It shows marked acute edema of the submucosa with very little inflammatory reaction except interstitial fibrin formation. The epithelium is in places denuded from the surface.

the columella. The area was tested with a sharp probe every 30 seconds till the pricking sensation disappeared. The paper was then removed and sensation tested every 30 seconds until it became normal again. Twenty patients were tested using pontocaine on one side of the septum and cocaine on the other, both at the same time.

In all but four patients the onset of anesthesia was more rapid with cocaine. The average time of onset was 3.9 minutes for pontocaine and 2.4 minutes for cocaine. The ranges were 1.5 to 4 minutes for cocaine and 2 to 7 minutes for pontocaine. The difference in speed of onset is significant ($p < .001$). The average duration of action was 7.6 minutes for pontocaine and 8.0 minutes for cocaine. The ranges were four to 12 minutes for each. Since the blotting papers were removed in each case as soon as the anesthesia was established, it follows that the time of application of cocaine was shorter than that of pontocaine. If the times of application had been equal it seems likely that the duration of action of cocaine would have been longer than that of pontocaine.

It may be concluded that 1 per cent pontocaine HCl has a potency either equal to or less than that of 5 per cent cocaine HCl, depending on what is considered a fair basis of comparison. In speed of onset the pontocaine was definitely inferior to cocaine solution in the strengths tested.

RESULTS OF QUESTIONNAIRE.

In choosing the clinics for our questionnaire it was felt that only the larger centers of the United States and Canada should be included. This was based on those clinics where a large volume of cases was to be found and where, also, the frequent use of these drugs would induce better technique in their application. Unfortunately some of the replies did not state, as requested, the number of cases treated and just said many thousands. These were discarded from the total of cases, but the complications were added so that these represent a group in a larger number of total cases and percentages cannot be estimated.

TOTAL NUMBER OF CASES TREATED, 39,278.
(With Cocaine and Pontocaine)

COMPARISON IN CASES WHICH USED COCAINE OR PONTOCAINE

Cocaine used exclusively	31,885 cases
Pontocaine used exclusively	7,394 cases
Total of cases reported	39,279 cases

CENTERS USING COCAINE IN TOPICAL ANESTHESIA
(Per Cent of Cases)

	%		%	
Boston	99.5	Philadelphia (1)	100	
U. Michigan	100	Philadelphia (2)	5	(allergic cases—bronchoscopy)
Vancouver	100			
U. Illinois	5	New Orleans	65	
Montreal	3	Toronto (1)	65	
Los Angeles	100	Toronto (2)	60	
		Chicago (2)	90	(uses both in each case with pontocaine spray first)

COMBINED USE OF ADRENALIN WITH PONTOCAINE OR COCAINE.

No conclusions or definite information could be recorded from the answer to this question. The amount used varied from equal parts to 1:4 in the nose and throat and a similar variance in the larynx and trachea. There is, however, a suggestion that the use of adrenalin might be an important factor in the faintness, rapid heart and vasomotor symptoms characteristic of the minor reactions. It is also a significant factor that several of the larger clinics, which use no vasoconstrictor drug, reported no reactions or deaths in their series of cases.

CONCENTRATION OF ANESTHESIA USED.

Cocaine.—Ten per cent in nose and pharynx, 5 per cent in larynx and trachea.

Pontocaine.—Two per cent in nose and pharynx, 1 per cent in larynx and trachea.

There were exceptions to this general rule. Most clinics insist that the quantity of pontocaine in trachea be limited to 2 cc. of 1 per cent solution. Various types of precautionary measures are used to assure the accurate measurement of this amount.

Untoward Reactions.—Under this heading it was our purpose to have a description of what was considered to be a reaction and we tried to classify these into three types: *a.* mild, *b.* severe (not fatal), *c.* deaths. The mild reactions were characterized by a fast thready pulse, fainting, sweating, pallor and other vasomotor symptoms. The severe reactions consisted of vascular collapse with changes in heart rhythm and convulsions. The deaths were due to cardiac failure or medullary failure.

CASES REPORTED WITH UNTOWARD REACTIONS

COCAINE		PONTOCAINE	
Mild	51 cases	Mild	36 cases
Severe	2 cases	Severe	7 cases
Deaths	3 cases	Deaths	4 cases

SUMMARY OF SURVEY.

Once more I must say that statistics are frequently misleading, but it is striking that in this large series of cases, from busy centers, that cocaine should be the anesthetic so generally used in preference to pontocaine. In view of this marked difference in the number of cases in which cocaine has been used it is quite significant that the deaths and severe reactions are more common with the use of pontocaine. There is little information gained from the rôle of adrenalin in combination with a topical anesthetic. A general agreement of the concentration of the anesthetic agent to be used in the various locations seems to exist.

Of timely interest is a survey made by Schindler[4] of 60 clinics doing gastroscopic examination in the United States, Great Britain and Australia. He reports on 22,351 gastroscopic examinations with only three deaths attributed to local anesthesia. All of these were from the use of pontocaine.

GENERAL SUMMARY.

Cocaine has been used and abused as a local anesthetic since 1884. It has been and is still used, in so many concentrations and by varying methods, that the accumulated knowledge of its good and also its dangerous effects has made it a relatively safe drug in experienced hands. We do know that experimentally it can be shown to produce topical anesthesia sooner and probably of longer duration than pontocaine. We have been shown by Leshure[5] and others that premedication with barbiturates reduces the toxic effects it can produce but does not preclude their appearance. We do know that individual hypersensitivity to the drug occurs so that small amounts, even as little as one-third gr. (*i.e.*, 20 mg.), according to Derbes,[6] may cause death. This hypersensitivity has been overemphasized according to our clinical survey. No satisfactory antidote is known to this type of reaction, although intravenous pentobarbital sodium relieves the convulsions and may control the toxic effect until the liver can perform its function of detoxication. According to Goodman and Gillman,[7] this occurs at the rate of one minimum lethal dose per hour.

It is not our purpose to discuss methods of safe use, but the suggestion of Whalen[8] that we have the patience to apply a small quantity locally by applicator or spray should help sort out, without a fatal termination, the very occasional hypersensitive individual. It has been pointed out by Titche that while scratch tests, patch tests or intracutaneous injections are satisfactory for drugs causing contact dermatitis, they are useless in drugs with other portals of entry. He concludes that when these deaths do occur with small doses, it is not due to an allergic response. Young[10] reports a remarkable case which they were able to follow by continuous electrocardiograph tracings during the progress of a fatal toxicosis. These showed auricular tachycardia followed by ventricular tachycardia and multiple ectopic ventricular beats before death.

There is some reason to believe that pontocaine, in the concentrations commonly used, is more irritating to the mucosal

tissues than cocaine. This is indicated by: *a.* the clinical impression that pontocaine is more irritating to the tracheobronchial mucosa of asthmatics and small children, *b.* more irritating to the cornea and conjunctiva, and *c.* by our animal experiments in which it proved to be much more damaging to the tracheal mucosa of the guinea pig and rabbit. In the concentrations in general use pontocaine certainly is not as effective as to the speed of onset of anesthesia and probably has not so long a duration period. The survey by questionnaire indicates that it is also not so safe to use in these concentrations as cocaine.

In this discussion no attempt has been made to assess the many reports in the literature of deaths due to pontocaine. Many of these have been made by internists in their use of this drug in the preparation for bronchograms. Our data was obtained from those clinics which were limited to otolaryngology or bronchoesophagology.

CONCLUSIONS.

1. An experimental and clinical study has been made of the local topical use of cocaine and tetracaine (pontocaine).

2. The mean fatal doses of cocaine and pontocaine were determined on guinea pigs by introduction of solutions slowly into the trachea. By this method the M. F. D. of cocaine was 24 mg./kg. and of pontocaine 7.2 mg./kg., giving a relative toxicity of pontocaine/cocaine of 3½/1. The usual clinical practice of using pontocaine in 1/5, the concentration of cocaine might be expected to give pontocaine a slight advantage in safety. This, however, was not confirmed by our clinical survey.

3. Clinical tests of cocaine 5 per cent and pontocaine 1 per cent by application to the nasal septum showed that the time of onset of anesthesia was about twice as long with pontocaine as with cocaine. The duration of anesthesia was about equal with the two drugs, although the time of application of the cocaine was shorter.

4. A survey of some of the larger clinics in the United States and Canada has been made with reference to the use if these two drugs with the complications encountered.

5. Cocaine is by far the majority choice in clinical use.

6. Complications of a severe nature and deaths were found more frequently with the use of pontocaine.

7. Cocaine has long years of useful service as a topical anesthesia and its vagaries are well known. It is still probably the most satisfactory of these two drugs to use in our specialty.

REFERENCES.

1. Bacharach, A. L., and Middleton, T. R.: The Toxicity of Amethocaine Hydrochloride (Pontocaine). *Quart. Jour. Pharm. and Pharmacol.*, 14:356-362, 1941.

2. Takahashi, K.: The Therapeutic Ratios of Some Local Anesthetics. *Proc. Jap. Pharmacol. Soc.*, 12:123-126, 1938.

3. Hamilton, H. S.; Westfall, B. A., and Ferguson, J. K. W.: A Comparison of Nine Local Anesthetics. *Jour. Pharmacol. and Exp. Therap.*, 94:299-307, 1948.

4. Schindler, Rudolf: Results of the Questionnaire on Fatalities in Gastroscopy. *Amer. Jour. Digest. Dis.*, 7:293, 1940.

5. Leshure, J.: Barbital as Preventive of Cocaine Toxicosis. *Anesth. and Analg.*, 6:189-191, Aug., 1927.

6. Derbes, U. J., and Engelhardt, H. T.: Deaths Following Use of Local Anesthetics in Transcricoid Therapy (Tetracaine); Critical Evaluation. *Jour. Lab. and Clin. Med.*, 29:478-482, May, 1944.

7. Goodman, L., and Gillman, A.: The Pharmacological Basis of Therapeutics. New York: MacMillan Co., 1941, p. 286.

8. Whalen, E. J.: Cocaine Anesthesia (Especially Cocaine and Pentothal, Barbital Derivative) in Peroral Endoscopy. *Ann. Otol., Rhin. and Laryngol.*, 53:469-479, Sept., 1944.

9. Titche, L. L.: Use of Tetracaine in Tracheobronchial Procedures. *Dis. Chest*, 13:452-455, Sept.-Oct., 1947.

10. Young, D., and Glauber, J. J.: Electrocardiographic Changes Resulting from Acute Intoxication (Cocaine). *Amer. Heart Jour.*, 34:272-279, Aug., 1947.

PSYCHOSOMATIC MEDICINE AND OTOLARYNGOLOGY.*†

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I propose then that the physician endeavor to find out by all possible ingenuity of conversation what matter of anxiety there may have been upon the mind of the patient that has rendered his life burdensome. Having discovered the burden, use all possible means to take it off. Offer him such thoughts as may be the best anodynes for his distressed mind... especially the right thoughts of the righteous and the means of obtaining composure of mind. Give him a prospect if you can of some deliverance from his distresses or some abatement of them.

Essays to Do Good... Cotton Mather.

It is probably an act of supererogation for a psychiatrist to appear before the Triological Society at this late date and to call attention to the fact that not only is our present-day concept of disease changing but that, also, disease itself is changing, for no specialty bears such eloquent testimony to this as does your own. Nevertheless, the things we have to say here need to be said at regular intervals, and if they prove unduly repetitious it cannot be helped, for, with Terence, we can only admit that "In fine, nothing is said now that has not been said before."

With the advent of the newer drugs and antibiotic agents, with the greater control of infectious diseases and epidemics and the greater increase in life span and life expectancy, the otolaryngologist, like other medical practitioners, finds himself confronted with an increased incidence of chronic disease

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and emotional illness, manifested in protean forms and testimony to the status of our present-day culture. It is this latter group which claims our attention today—the emotionally distressed—as they present themselves in the offices of practitioners in the various fields represented here; but it would be well for you on another occasion to consider the emotional reactions of members of the older age groups, for before long a large segment of your practices will be concerned with this forgotten and neglected element in our medical population.

It is reasonable to suppose that the practitioners of your specialty see a large number of emotionally disturbed persons, for you work in an area important not only because of the special senses involved but also for cosmetic reasons. This is true because part of your field encompasses the facade of the individual which, attractive or not, is presented to his fellow man with varied emotions. My contention in this thesis is that you as specialists can do much to aid these distressed individuals, with their variety of psychosomatic complaints, and it has the added corollary that unless you do undertake the treatment of the milder conditions you encounter, many of them either will not be treated at all or, worse yet, will be treated by someone in a manner which, no matter how well intentioned, will serve only to fix the neuroses *in situ*.

There is no need for me to list the conditions in question; you know them well: the people whose noses are too short or too long, too thick or too thin, too wide or too narrow; those who are dizzy or who have "sinus," who have lumps in their throats or whose heads ache with varying intensity; the asthmatic, the "sneezers," those with coughs and allergies and all those who plague you by presenting symptoms enough but no organic basis to justify their complaints. You are better acquainted with them than am I, yet we should consider them together.

It has been said of medical specialists that their function is to lessen their importance as specialists and to strive to have their teachings incorporated into the body of medical practice. This effort is particularly necessary in the practice

of psychiatry today, for there is general agreement among all concerned that even though psychiatrists are charged with the care of neurotic patients, most of the milder psychosomatic conditions will have to be treated by the practitioner or the specialist in whose field they fall, not only because they are in a much more favorable position to do so, but also because there will not be enough psychiatrists to assume this tremendous burden, even if that were desirable.

The thought might enter your minds that the already harassed otolaryngologist does not want this type of patient or practice, but the answer to this is—he already has them. Like God's poor, they are in plentiful supply, and after the gastroenterologist, it is probable that the otolaryngologist sees a larger number of them than does any other specialist. Some of these patients exhibit the frank neuroses and even the psychoses, while in others the psychiatric tones are subtly disguised. Many of them are "just nervous," but all of them are in need of help. Simply to designate their complaints as functional and to tell them to "go home; the troubles are all in your head" will not suffice—this is comparable to the psychiatrist telling them to go on a vacation, the trouble is all in their bodies. Either they need help from the practitioner they consult medically or they need to be put in the hands of someone who can understand and treat their ills.

Mrs. W., age 50 years, is an excellent example of the psychotic group mentioned above. For the last 10 years she has been making the rounds of otolaryngologists, dentists and oral surgeons. Her complaints are "pain in the mouth, tongue, cheeks"; "in the flesh" as she puts it. From physician to clinic, to dentist and surgeon she has made weary rounds. Most of them have assured her that it is "all due to her nerves" and have passed her on.

Very gentle and careful questioning reveals that under these complaints is an involved delusional system. She regards these pains as "just visitations" upon her. Actually, she believes they are all her husband's fault. Her delusional system includes people following her; people watching her; people asking questions which have multiple meanings; people talking about her; husband trying to kill her, and so on.

This patient has paranoid schizophrenia. It is useless to talk to her about her ideas being delusional. She counters by saying, "It can't be true; otherwise those other doctors would have told me so." The whole operation of diagnosing this illness takes but a few moments. Detected early, she would have been a candidate for insulin therapy; now it is probable that she will be a suicide, because she has given up hope and is depressed.

Mrs. X is not psychotic, but she does have a crippling neurosis. She is 36, an attractive person and her main complaints are "stiffness of the neck, tightness of the throat and difficulty in swallowing." She dates her illness to two years before her visit; it began the day her father died. She had been "his girl." Actually, she has been ill much longer than she suspected.

Careful questioning indicates that she has some insight into the causes of her illness. Her real trouble is a terrific antipathy to her mother. She is constantly fearful that she will betray her hostility; she is constantly holding herself in. No wonder she has stiffness of the neck and tightness of the throat.

As an only child, she was her father's pet. She was more at home with older people and could not compete with girls her age. She now looks down upon her friends and neighbors and feels superior to them. She remarks, "I am very ambitious." The relationship between her difficulties and her symptoms is obvious here, and even the patient recognizes it.

The emotional reactions of human beings are so involved in their symptomatology that the time has come when the physician can ill afford to know less about them than does the modern layman, and the layman's knowledge of the subject has greatly increased since the outbreak of World War II, for he can hardly go to the cinema or read a novel without encountering a psychiatric problem. A great deal has been learned in the last several decades from the scientific investigation of human nature, and some acquaintance with the more basic findings of psychosomatic medicine is indispensable for every practicing physician.

What is psychosomatic medicine? Even to mention the subject usually has been reason enough to start a fracas at almost any medical meeting. It is probable that one of the best definitions of what it is and what it implies is to be found in the journal, *Psychosomatic Medicine*,[1] in the introductory statement to its first issue. After noting that psychiatry's principal interest is the diseased mind, the statement declares that psychosomatic medicine covers a broader field and a different one. Its object is to study in their inter-relationships the psychologic and physiologic aspects of all normal and abnormal bodily functions and thus to integrate somatic therapy and psychotherapy. It designates a method of approach to the problems of etiology and therapy, rather than a delineation of the area. Briefly, it concerns itself with the

psychologic approach to general medicine. "It is both a special field and an integral part of every medical specialty."

There are, of course, many misconceptions concerning psychosomatic medicine, and this is particularly true in its relationship to otolaryngology. To some psychiatrists, unfortunately, it has meant an undue emphasis upon *psyche* and not enough upon *soma*. To some otolaryngologists, the reverse has been true. Actually the concept means only to give each factor its rightful proportion and place. It is thus a point of view and a method of approach.

To utilize the psychosomatic approach, several things must be accepted as axiomatic. The first is that every disease has a psychic as well as a somatic component, and it is, therefore, necessary to know what sort of person it is who has the disease as well as what sort of disease the patient has. Second, we must regard disease as a process and not as a fixture or a series of end-results. Every disease process, whether it be designated "mental" or "physical," goes through a reversible or a physiologic change before it becomes structuralized and irreversible; therefore, the sharp distinction supposedly existing between functional and organic is extremely nebulous, for there is no one to say when functional disease ends and organic disease begins. A good example of this statement is cardiospasm. McMahon, Moersch and I[2] recently reported upon a group of 25 patients who had cardiospasm and who in addition to the necessary physical treatment underwent rather complete mental examinations. Twenty-four of them had suffered from emotional trauma at the time of the onset of their symptoms, and recurrences were noted after repeated or new psychic trauma. In general, the symptoms became aggravated when the patient was upset or "nervous." A number of environmental and emotional factors were noted to be present at the time the illness began. These included hostility and resentment, economic loss and insecurity, death of a parent on whom the patient was dependent, feelings of rejection, fears of death, insanity, failure and even surgical procedures.

As a group, these individuals gave evidence of personality difficulties in childhood plus neurotic difficulties in adult life. These findings bear out the contention that cardiospasm is a disease of the frustrated, and is not seen in happy, contented or well adjusted individuals. Yet, it is obvious that it would be difficult to tell when physiologic changes ended and structuralization began in their cases.

Third, we must include emotions in our list of potentially pathogenic agents, noting that they may become such under certain conditions and that when these conditions occur, conflict is set up. This conflict may be so painful that it is excluded from consciousness. This should not be new to us, for it was described by the physiologists, Pavlov and Cannon. John Hunter demonstrated it graphically when he made his famous declaration that his life was in the hands of the rascal who made him angry, and then proved it later by dying in a fit of rage.

Fourth, we must be willing to admit that a potentially injurious agent, be it emotional or bacterial, cannot bring about disease unless the organism is prepared for it in a manner physiologic, psychic, or both. Experiment has shown that emotional stress seriously limits the ability of the organism to regain a stable equilibrium after it has been subjected to some strain or injury.

If we are willing to accept these axioms, then the next step requires us to make an effort to understand the individual and the manner in which he reacts to stress. To paraphrase Dr. Alan Gregg—we must not consider the personality of an individual to be merely an inconvenient appendage to the particular organ we are studying. To understand people, we must be willing to sit patiently and to listen to them, and while we are listening to what they are saying, we must surmise what it is they are not saying and to judge how it is affecting them. We must expect our advice to be eagerly sought and then not followed; yet, in the face of this and numerous other frustrations we are required to exhibit that attitude of equanimity which Sir William Osler held to be one of our essential medical virtues.

This concept of the physician-patient relationship is no psychiatric mumbo-jumbo. It is one of the most essential ingredients of every therapeutic process. We cannot blithely dismiss it as mere bedside manner, for in every meeting of the physician and patient there is either a psychotherapeutic or a psychonoxious influence at work. From the initial interviews until the last meeting, in the physical examination and in the prescription of drugs, emotional and other cross-currents are playing a rôle. Upon a physician's ability to direct these cross-currents hinges a large part of his success.

At times, patients tend to be overdependent upon physicians and at other times to seem hostile or belligerent. The doctor's task is not to respond to either of these attitudes, for they are not meant for him personally. They are directed at what the physician stands for in the mind of the patient. Sometimes the patient tends to regard the doctor as a minor deity, but this is not harmful unless the physician develops the belief that this attitude is correct and justifiable. Then his usefulness to the patient is at an end. At other times, when the patient is hostile, the hostility is not a personal thing either, and to respond to it by becoming angry removes the possibility of helping him. The only time a physician becomes angry at a patient is when he, himself, is frustrated, and he should understand that patients' reactions to him usually are conditioned by their relationship to authoritarian figures early in their lives, especially parents and teachers.

In addition to what the patient thinks of his own illness, what he thinks of the physician who treats it also is important. In regard to the former, one thing is certain: the patient rarely is objective about his illness. He presents anxieties, fears and physical symptoms. He wants, or thinks he wants, treatment and whatever help is available. He may readily admit he is nervous or in fact that the whole family is nervous, but he fails to equate these facts with his presenting symptoms. While the whole situation may be obvious to the physician who can readily relate psychic and somatic elements, the patient does not see the connection or if he does

see it, he sees it intellectually without feeling it. These problems which he has disguised as "symptoms" are sources of deep and painful anxiety to him.

Mr. Y is an example of this type of reaction. Twenty-eight years old, and unmarried, he had been a regular visitor to the out-patient department of a large city hospital. His chief complaints were headache, which he insisted was due to "sinus," and some abdominal pain. Although he was studied carefully, no organic disease was found. The symptoms did not respond to treatment, and had progressed to the point where the headache now interfered with his work. This man's illness had begun four years previously. When he was first questioned regarding any possibility of trouble at the time of the onset of his symptoms, he stated that he had been much excited and upset about a personal situation, but he did not see why he should go into that, for it had nothing to do with his headache and abdominal pain. Further questioning brought out the following facts:

Four years previously the patient had been haled into court along with several other young men in his neighborhood. One of the young ladies in his locality had borne a child, and its paternity was to be decided in the municipal court. Over his violent protestations, he had been adjudged the winner of the contest, and along with the doubtful honor of fatherhood which was conferred upon him, he also was awarded the privilege of supporting the child until it was 16 years of age.

In the ensuing period of emotional turmoil he commenced to have difficulty with his fellow employees and also to hate his job. He was now in the anomalous position of making money in a job which he hated, and paying part of it to a girl he hated even more. He was in a dilemma. He could not run away; he was under court order and, in addition, was the only support of a widowed mother.

One other problem arose to plague him. In an unguarded moment he had discussed matrimony with another young lady, and she was now busily engaged in making plans for the wedding. His ideas on this subject had changed, but he said he did not want to hurt her, so he guessed he would have to go through with it.

Although the patient insisted that the problems which he related had no connection with his headache and abdominal pain, the relationship is obvious. Furthermore, he appeared to have enough difficulty to keep him supplied with headaches until the child reached the age of 16. It is also obvious that illness might offer a way out of his unpleasant situation.

The solution, from a psychiatric standpoint, might come about as follows: *1.* If John is ill he cannot work; this takes him away from an unpleasant job. *2.* If John cannot work, he receives no pay and it is impossible for him to support a child, despite the court order. *3.* If John is sick and cannot work, it obviously is unfair for him to marry and expect his wife to work and support him. Thus, the third unpleasant possibility facing him is removed.

No amount of local treatment would have cured him of his headache. The above mentioned method of approach to the problem, admittedly superficial, is within the range of every practitioner.

An important thing for us to keep in mind, in regard to the foregoing patients and in fact in all patients, is that their neuroses have meanings. They do not develop neuroses simply to annoy or harass physicians. They follow a purposive pattern in their symptoms, and this pattern represents the individual's reaction to the situation within himself or the surroundings which he finds difficult and unmanageable. His method of reacting to situations is his very own, and it is determined by his personality and by his previous experience. His reactions represent a compromise between the different arguments of his personality or between his personality and environmental influences. Ideas which become too painful are repressed, but the tensions they engender remain. When the stress of these varied emotions becomes too great, they appear in the form of symptoms, often in symbolic form, and hence the patient's neurotic manifestations frequently give a clue to the underlying difficulty.

It is evident, then, that a number of patients who come to the otolaryngologist for diagnosis and treatment present symptoms which are nothing more than the regular physiologic manifestations of anxiety. With the inevitable tendency toward self-diagnosis, these symptoms may be formulated by the patient into "sinus trouble" or "dizziness" and when, through introspection and suggestion, such a patient has added a few layers of his own ideas of what a sufferer with these diseases should have, he presents a complicated problem. During the examination one has an opportunity to observe the moist palms, the tremors, the wide pupils, the perspiration and the general restlessness which betrays the presence of anxiety. Miss Z typifies members of this group.

The patient is 39 years old; she was divorced after an unfortunate and short-lived marriage. Her complaints are of tinnitus and dizziness. Careful physical, neurologic and otologic examinations fail to reveal organic disease, but all consultants agree that she probably had Ménière's syndrome at the inception of the illness. The patient obviously is under stress and shows all the symptoms of anxiety mentioned above. Upon questioning, it develops that her husband was uncouth and brutal, and that the patient was pushed into the marriage by family pressures. The husband left her after infecting her with a venereal disease. Upon her return home the patient withdrew from her friends and society, fearful that people were critical of her. She had resisted attempts of her husband

to initiate her into abnormal sexual practices. All this was the proximal background upon which the neurosis could develop. The remote background was an insecure childhood in a foreign settlement. The exciting cause of the emotional upset was the overhearing of a conversation through the consulting room door in a physician's office. From this conversation the patient got the idea that perhaps she was abnormal sexually. At approximately the same time, a man in the neighborhood was arrested for molesting children. The patient made an unfortunate connection in her mind, and compared this man with herself. All her old insecurities were mobilized, and the patient feared to leave the house lest she be pointed out as abnormal. Her first attacks of dizziness occurred at this time, and she began the rounds of physicians, seeking help. As her anxiety increased, her dizziness became worse and her illness was soon in a vicious cycle. After several interviews and an understanding of her fears and panic, her convalescence began.

The situation typified above and also admittedly oversimplified, gives an indication of the involved psychopathology which must underlie and accentuate the ordinary complaints which patients bring to us, and which exist in the absence of any organic pathologic process. The focus of these difficulties was determined by the original slight infectious process. To seek out the causes of psychosomatic problems, we need the answers to three questions:

1. What were the original characteristics of the personality of the patients in question?

2. Why did they become ill at the particular time they did?

3. Why did they become ill in the manner in which they did?

If we know the answers to these three questions, we shall have taken the first step toward understanding psychosomatic manifestations and, as someone has indicated, the only illnesses which are not psychosomatic are those which lie in the realm of veterinary medicine. It is seldom, however, that we obtain the answers to these questions in a short or superficial examination. The answers must be obtained by gentle, careful and thorough investigations into the psychologic background of the individual. Any attempt to badger the patient to produce satisfactory answers is foredoomed to failure.

As mentioned at the beginning of this discussion, all that one can hope to accomplish in a resumé of this type is to renew the interest of the otolaryngologist in the psychosomatic approach to problems, and to bring some of its elements into sharper focus. This leaves the whole question of treatment unanswered, but fortunately the various available psychotherapeutic methods are now outlined and simplified in numerous publications intended for the general practitioner. The main thing to keep in mind is avoidance of meddlesome psychotherapy and the stirring up of anxiety which is latent. No one ever got into trouble of this kind by listening patiently, questioning carefully and formulating the problem simply and with understanding in his own mind.

The question of symptomatic treatment was mentioned in the discussion of the illustrative cases. Lest there be some misunderstanding and these allusions be thought to be critical, it should be noted that we recognize symptomatic treatment not only to be justifiable, but in some situations undoubtedly to be the treatment of choice. Not only does it enable the physician to alleviate troublesome symptoms, but at times it acts against the basic difficulty itself. In some cases it breaks up the vicious cycle in which the pathologic process conditions certain alterations, which alterations in turn further the progress of the disease. Although this approach admittedly is not curative, it may restore a balance which influences favorably the patient's feeling, tone and capacity to work.

Not even in the use of the new hormones, ACTH and cortisone, in the treatment of the various otolaryngologic conditions for which they might be indicated, can you afford to forget the psychosomatic approach, for the administration of these hormones frequently has been associated with subjective and objective psychologic changes. Rome and I[3] have graded these changes as follows:

Grade 1 includes the improved emotional tone as expressed in the responses of those who express their relief in their speech and behavior. It seems in some to be the direct consequence of a rejuvenated hope.

Grade 2 includes the responses of patients who showed a quantitative elaboration of this reaction. They are greatly stimulated in their thinking and behavior, and in some instances are accelerated to the point of mental excitement, restlessness and a rapidly fluctuant mood. During periods of elation they tend to be facetious and even silly.

Grade 3 is comprised of the responses of patients whose personalities before the administration of cortisone or ACTH showed evidence of serious psychologic conflict which was poorly managed. Within a short time after the administration of the hormones, these conflicts were fulminated, and the physician then had a disturbed individual on his hands.

Grade 4 is a relatively unusual response. The clinical picture is an overtly psychotic one of unusually brief duration. Its character is determined by the patient's prepsychotic personality.

These psychologic changes, all of them relatively short-lived and all of them reversible, probably are due to the sudden and profound alterations, the *milieu intérieur*, caused by the hormones and the consequent effect upon the patient's metabolic homeostasis. The variety and degree of this reaction are due to many causes, and collectively they constitute a major stress, as taxing of his psychologic adaptation as any serious external stress would be to an equally vulnerable person; therefore, the possibilities of untoward reactions will have to be kept in mind when these hormones are administered.

There are many other facets to the psychosomatic problem, for we cannot practice medicine in a vacuum. Before long we shall have to begin to pay more attention to the *milieu* in which the *psyche* and *soma* operate, and this will necessitate sociologic considerations. This has nothing to do with social medicine; it means simply that the individual is strongly influenced by his environment and social surroundings.

This is never more patent than it is in military medical practice, in which some of the younger otolaryngologists will soon find themselves. I am convinced that the touchstone of

military psychosomatic conditions is in motivation and morale. It makes little or no difference in many of these patients how severe is the neurosis from which they suffer if they are properly motivated. The cure of their psychosomatic symptoms, therefore, lies in prevention of these symptoms, and this prevention lies in proper motivation. This proper motivation should be developed in our high schools and our colleges, but to go into that would take us too far afield.

One cannot see the things we have spoken about here today through a head mirror. They are the larger aspects of the problem, and yet they vitally concern each one of us here, for they speak of factors which influence our patients. In the services they influence our fighting men, and the importance of keeping the latter group in good health becomes more apparent every day.

Now, somewhat fearful that I have meandered about and tread injudiciously upon medical toes, I desire to offer an olive branch. If I needed your ministrations, I would not want you to retalitate by offering to engage me in sociologic discourse. I close this dissertation in the words of Burton, who in his "Anatomy of Melancholy," also became perhaps justifiably timorous, being fearful of the future treatment he might get. He said: "I will urge these cavilling and contumelious arguments no further lest some physician should mistake me and deny me physic when I am sick."

For my part, I assure you I am well persuaded of physic and of the otolaryngologist's art

REFERENCES.

1. Editors: Introductory Statement. *Psychosom. Med.*, 1:3-5, Jan., 1939.

2. McMahon, J. M.; Braceland, F. J., and Moersch, H. J.: The Psychosomatic Aspects of Cardiospasm. *Ann. Int. Med.*, 34:608-631, Mar., 1951.

3. Rome, H. P., and Braceland, F. J.: The Use of Cortisone and ACTH in Certain Diseases; Psychiatric Aspects. *Proc. Staff Meet., Mayo Clinic*, 25:495-497, Aug. 16, 1950.

SECRETORY EFFUSION OF THE TYMPANUM.*

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The title of this paper has been chosen because it suggests a sterile secretion in the middle ear or mastoid cells. Any one or both portions of this system are frequently involved.

Various terms have been used to describe this condition but perhaps the ones most commonly mentioned are hydrops of the middle ear and secretory or catarrhal otitis media. The two latter terms give the impression of an inflammatory process in the middle ear, and while a mild inflammation often initiates the condition the fluid behind the drum is almost invariably sterile.

Although a number of articles have appeared in recent years on "Secretory Otitis Media" the condition was recognized and accurately described by Politzer about 80 years ago. Quoting from one of Politzer's students — "In secretory catarrh the liquid is often brownish in color and this gives a yellowish tinge to the drum. The membrane frequently appears shiny as though the patient instilled a little oil in his ear. The drum is often infiltrated with fluid."[1]

About the same time that Politzer[2] described exudative catarrh of the middle ear, Sir William Wilde wrote his book on "Aural Surgery." An Irish pioneer in otology, Wilde made many observations but is perhaps best remembered for his classical "Wilde's incision" for acute mastoiditis. Although he carefully described the incision behind the ear, he never actually opened the mastoid cells. According to Stevenson and Guthrie,[3] "Wilde did not practice paracentesis for acute

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otitis, but in cases of so-called Eustachian obstruction he made an artificial perforation of the tympanic membrane, cauterizng the edges with nitrate of silver to ensure patency."

The apparent prevalence of this condition, as shown by the number of recent articles,[4-11] is in all probability due to the widespread use of chemotherapy. Since the advent of the sulfacompounds in the middle thirties and penicillin in the early forties, undoubtedly many cases of acute nasopharyngitis have been checked which otherwise would have developed suppurative otitis media. It has also been suggested that virus infections are often responsible. The increased popularity of plane travel has also shown a greater number of cases of aerotitis, sometimes with effusion. As a result patients frequently are considered cured, who still have a residual exudate in the middle ear which is free of organisms. The fluid may remain for days or months and varies in amount in different patients. Under the care of an able otologist this condition should not be allowed to persist.

Geographically, secretory catarrh does not appear to be limited to any particular area; it is widely distributed and found in all ages. Climatic conditions seem to have little bearing on its frequency, although during the Fall and again in the Spring, when changeable weather is the rule, acute upper respiratory infections are more prevalent, and effusion into the tympanum seen more often.

Etiology: An inflammation of the nasopharynx producing obstruction of the Eustachian tube is the most frequent cause of effusion into the middle ear. The swelling may be limited to the beginning of the tube or involve the whole tube and tympanum as mentioned above. The chief causes of this condition are so well known that repetition is unnecessary. They can all be summed up under the term obstructed noses. Whether the nasal obstruction is mechanical in origin or due to infection or allergy makes little difference. The result in the tympanum is identical. Two important causes in adults which should always be kept in mind are malocclusion of the teeth and nasopharyngeal growths.

The importance of tumors of the nasopharynx as a cause of effusion into the tympanum cannot be emphasized too strongly. During the preparation of this paper a case presented itself which gave a typical history.

A married woman of 46 years complained of a blocked sensation in the left ear. This symptom had been present for the previous six months and she had been treated elsewhere, the tube being inflated 18 or 20 times. The hearing returned to normal after each inflation, but this temporary improvement lasted only for a day or two. The left drum was markedly retracted and showed a dull reddish color—the right drum being normal. Inspection of the nasopharynx with the nasopharyngoscope and postnasal mirror showed a roughening of the tissue around the Eustachian tube as compared to the normal tubal opening on the right. A biopsy under local anesthesia proved to be squamous celled carcinoma. X-ray films of the base of the skull showed marked erosion of the basisphenoid. The patient was given a series of X-ray treatments but one cannot help but feel that valuable time was lost. In relating this case two facts should be stressed. The catarrhal deafness which recurred within a day or so after inflation should have made the otologist suspicious. The very nature of the temporary improvement should at once have prompted a thorough examination of the nasopharynx. This neglected area should, of course, be investigated with all the means at our disposal—if necessary, after cocainization. As a general rule cervical adenitis is the earliest complaint in nasopharyngeal tumors,[12] but fullness in the ear with conductive deafness, otalgia and tinnitus which persist should also be kept in mind as possible symptoms.

Although effusion into the tympanum was known and described in great detail well over a half century ago, a few practical points might be mentioned that have not been noted in recent articles.

Pathology: According to Prof. Ruttin, of Vienna, who was a pupil of Adam Politzer, catarrhs of the middle ear are divided into *A.* acute catarrh, *i.e.*, *1.* dry, *2.* moist, and *3.* secretory catarrh; and *B.* chronic catarrh. "They are characterized anatomically by only slight hyperemia, swelling and tumefaction of the mucous membrane, and by the extravasation of a clear, serous, or viscid, sticky mucus exudate into the tympanum."

In dry catarrh, the drum membrane is retracted, but there is no secretion in the tympanum or even in the mucous membrane lining the cavity. The mucosa may appear quite normal.

In moist catarrh, there is secretion in the mucous membrane, but no fluid in the tympanum. The lining mucous membrane is swollen and edematous.

If the process goes on to the stage of secretory catarrh, free fluid appears in the tympanum. As mentioned previously, the fluid is either thin and serous in consistency or thick and viscid. The thin serous effusion, which is much more common, is thought to be due to negative pressure and is more in the nature of a sterile transudate.

The thick, sticky mucous secretion is more of an exudate and varies in consistency depending upon the amount of mucus present. Serous fluid has a high protein content which coagulates readily when exposed to the air, while mucus fluid has a low protein content. In both cases the fluid is sterile.

According to Ruttin, dry catarrh is found in cases of slight colds, especially when the anterior parts of the nose are affected. In such cases the drum membrane is grayish and retracted. Moist catarrh results from inflammation in the posterior parts of the nose and nasopharynx, such as occurs in children with enlarged tonsils and adenoids and in adults with posterior tip hypertrophies or swollen lateral bands. In this type, the drum markedly retracted often shows a reddish tinge, due to swollen mucous membrane shining through. The retraction is thought to be the result of a mild inflammation of the tensor tympani muscle which pulls the membrane inward.

Often in cases of secretory catarrh where the Eustachian tube is closed, the process is not limited to the tube but also involves the middle ear and all connecting mastoid cells.

Symptoms: The patient frequently complains of a blocked sensation in the affected ear with dullness in hearing and sometimes a lack of resonance to the voice. There is usually a history of a previous cold, and tinnitus is common.

Often the patient says his voice sounds as though he were "talking in a barrel." The degree of deafness and fullness depends upon the position and amount of secretion present. A patient often volunteers the statement that the hearing is dull while lying in bed and improves on assuming the upright position. In the recumbent position the oval and round win-

dows are covered, impairing the hearing; but on arising the fluid sinks to the bottom of the tympanum, and the hearing improves. At times patients complain of crackles in the ear due to bubbles of air in the fluid.

Frequently both ears are affected. Although occasionally symptoms are indefinite or perhaps lacking altogether, every experienced otologist should be able to diagnose and treat these cases.

Signs: The appearance of the drum varies, depending upon its thickness or transparency, the amount and color of the exudate, duration of the course, and the degree of reddening of the tympanic lining membrane. When enough secretion is present to show a fluid line or meniscus across the drum the diagnosis is relatively simple.

The variations in color and position of the fluid level are well known and have been described frequently. An exudate which gives a yellowish or amber color to the drum below the fluid level is as a rule thin and serous, rather than heavy and viscid.

One or two other observations of Ruttin are worth mentioning: "When the fluid level line is below the umbo of the malleus it usually goes across the entire drum membrane. When it is above the end of the handle it generally goes half way. If in doubt about the fluid level always inflate the tubes or move the head from side to side to change the level line. When the hearing is not improved by air inflation, the case is not secretory catarrh."

There are exceptions, of course, when air inflations do not appear to improve the hearing and in these occasional cases one is justified in doing a paracentesis. It is much safer to incise the drum when in doubt than to leave secretion in a tympanum which may later result in bands and strands of fibrous tissue binding down the ossicles and producing adhesive deafness in later years.

This is particularly true in children. The cases most frequently undiagnosed are those in which the tympanum and

perhaps the mastoid cells are filled with fluid. In the absence of a fluid line one has to depend upon the color of the drum to diagnose the presence of secretion. The range of colors has been variously described as yellow, amber, greenish-yellow or red, sometimes verging on a reddish purple. When the yellow or amber color is seen, it is more marked behind the umbo, *i.e.*, over the promontory. Often in these cases a fluid line will appear after air inflation. When the drum is one of the above shades the color is well marked with more luster than usual and the retracted handle of the malleus is narrower and very clearly defined. (In the normal malleus, two-thirds of the thickness of the handle is connective tissue, and the fluid behind the drum makes this tissue transparent. This accounts for the narrow appearance of the handle.)

Auscultation is often an aid in making a diagnosis. To those accustomed to using the Eustachian catheter the sound of air through the tube is very characteristic. Any abnormal sound, whether it is a crackling noise, bubbles, a series of squeaks or a faint thud, should make one suspicious of fluid in the middle ear. If the drum before inflation or catheterization appears thickened and retracted with a very prominent short process, the diagnosis is more definite.

A Siegle's speculum is also of value. When suction is applied in the external meatus by means of this instrument the hearing is often improved. Sometimes a fluid level appears which was not present before using suction. As mentioned, if there is any question of fluid in the tympanum after a detailed history and careful inspection an opening should be made in the drum. Whether this is done by means of a hypodermic needle or a paracentesis knife is a matter of choice.

A No. 22 or perhaps a larger No. 20 needle, two and one-half to three inches in length, with a short bevel of 1 to 1.5 mm., is usually sufficient. Many able otologists object to this method, as the opening often tends to close too quickly and has to be repeated. They prefer to incise the drum to allow

even viscid fluid to exude more freely. There is a marked difference of opinion as to where the incision should be made. This will be dealt with under Treatment.

In most cases the drum can be incised without anesthesia, even in young children. The objection has been raised that a local anesthetic on the drum increases the chance of infecting the middle ear. This has not been borne out by experience. Unless the knife touches the promontory on the inner wall there is little discomfort. This has been mentioned previously many times.

A preliminary inflation of the tube raises the intratympanic pressure so that on opening the drum the contained fluid is often forced into the meatus.

Most otologists seem to prefer inflation and spot suction to the use of the Siegle's speculum. Applying suction directly to the opening in the drum instead of the whole canal causes less discomfort and seems to be more effective. A pledget of absorbent loosely placed against the drum before inflation or catheterizatnon tends to prevent the fluid from being drawn back into the tympanum.

Prognosis: The outlook depends upon the duration — whether days or months, the consistency of the secretion and the form of treatment instituted. As a general rule the earlier these cases are seen the better the prognosis and the less fluid present the quicker it tends to disappear.

When the symptoms have been present for only a few days, simple inflations clear up the vast majority of cases. On the other hand, if fluid has remained for months or years, often persistent treatment over a long period is required. In obstinate cases the drum may have to be opened many times before the condition finally clears.

Any associated nasopharyngitis should, of course, receive early attention. In children adenoidectomy is often indicated, and occasionally in adults the removal of enlarged posterior tips of the inferior turbinates is necessary.

Persistent nasal obstruction should be corrected and hypertrophied lymphoid tissue or enlarged lateral bands treated. After any of these operations it is wise to wait at least two weeks before inflating the tubes. Allergic factors must be attended to and the possibility of endocrine disturbances kept in mind.

Treatment: According to the late Prof. Alexander, who was a contemporary of Adam Politzer, attention should first be directed to the nose. Any infection in the upper respiratory tract, particularly, the sinuses, should be corrected. In treating the affected ear air inflations are carried out. When patients are seen early simple politzerization is usually sufficient, but in old chronic cases often the catheter is necessary. Usually air inflation lasts only a few days, so this should be repeated every second or third day while the nose is treated at the same time by some vasoconstrictor. Often a change of atmosphere helps, such as the dry air of the mountains or perhaps the seashore. Sometimes allowing a patient to use the Politzer bulb himself, providing there is no acute coryza present, will hasten his recovery. Alexander frequently carried out this procedure for a month before sending a patient away. Of course, in these days this is not always practical.

Air inflation acts best when the fluid is serous and is not nearly so effective when heavy mucus is present. If the head is bent forward and tilted so that the affected ear is uppermost, inflation tends to allow the fluid to drain away through the tube more easily. In chronic cases the results are better if the patient sucks in air while inflation is carried out. Sometimes during inflation patients complain of marked dizziness. This is due to increased pressure against the round window. Alexander insisted that the catheter be reserved for chronic cases as the mucous membrane has already been injured.

When the hearing is not improved after the successful use of the Politzer bag it is wise to incise the drum. There is a marked difference of opinion as to where the incision should be made. Some writers prefer the posterior inferior quadrant as it is further away from the inner tympanic wall.

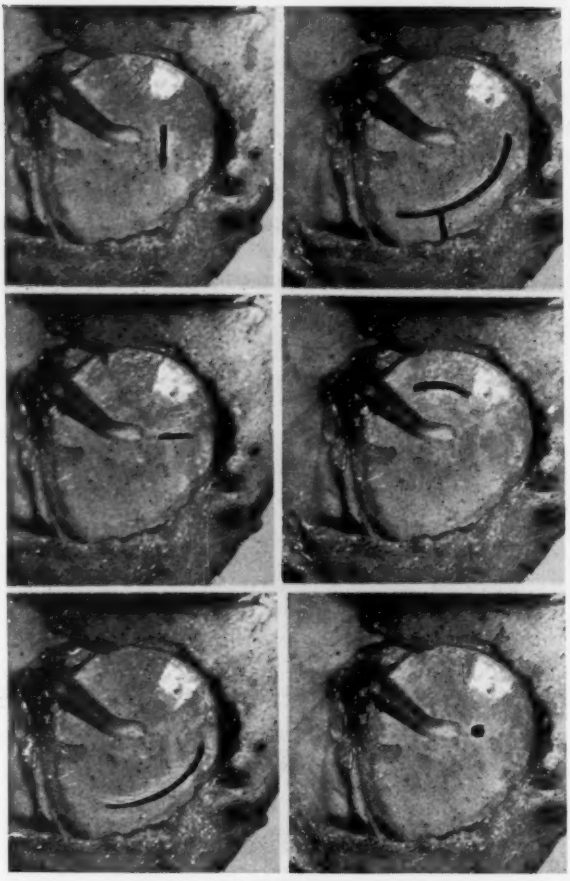

Fig. 1.

Others make a vertical incision just below the umbo. Still others suggest a horizontal incision below the end of the handle of the malleus, so that the floor of the external meatus is not injured. A favorite site for those who use a large hypodermic needle is just below the umbo at the six o'clock position.

In general it is well to remember that if there is pressure behind the drum the opening should be made in the most bulged part. At times it is necessary to make a second opening, especially if the secretion is of the heavy mucoid type. It has been suggested in recent years that this counter-opening be made in the upper anterior quadrant.

In the opinion of the author the initial incision should be made through the light reflex, *i.e.*, in the anterior inferior quadrant. The radiating fibres are the strongest in this area and as a result they tend to keep the incision open longer.

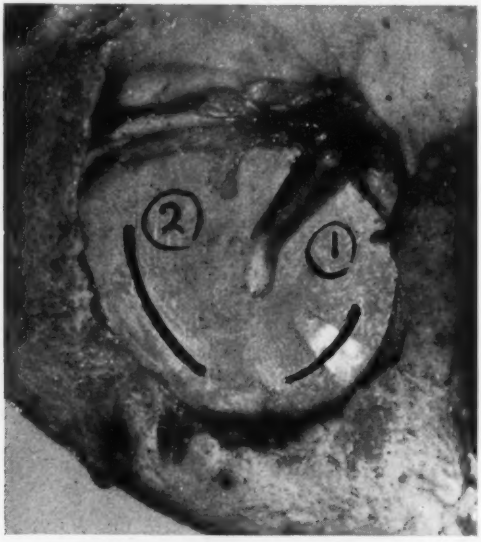

Fig. 2.

If myringotomy is performed anywhere else the opening usually closes within 24 to 36 hours. It is also inadvisable to make a large horseshoe incision as there is liable to be marked retraction and scarring.

The tough radiating fibres in the lower anterior quadrant have nothing to do with the cone of light. It is just a coinci-

dence that they happen to be present in this region. After paracentesis air inflation should be carried out and spot suction. If no secretion appears and the hearing is not improved a second incision is recommended at once. This is made posteriorly, in the usual site for purulent otitis media. Often heavy mucus secretion becomes walled off, and this counter-opening is advisable.

Again after myringotomy repeated air inflations are often necessary, followed at times by spot suction or rarefaction by means of a Siegle's speculum.

When treated in this way over one-third (nearly 35 per cent) of cases of secretory catarrh clear up entirely without any after effects or repeated myringotomies. As children are commonly affected this is important. Occasionally repeated paracenteses are necessary.

Myringotomy should be performed without anesthesia and without swabbing the external canal with alcohol. Using a sterile paracentesis knife there is little danger of infection, and the discomfort can be compared to the momentary twinge of a hypodermic needle in the skin. On the other hand, a pledget of absorbent moistened in Bonain's solution (menthol, carbolic and cocaine in equal parts) placed against the drum in apprehensive patients does not seem to increase the chance of infection.

When indicated, myringotomy affords great relief, but this alone is not sufficient, unless contributing factors are corrected. Although the details and sites of paracenteses have been described, we should always bear in mind that almost 75 per cent of cases of secretory catarrh respond to conservative treatment. Relatively few in comparison require myringotomy in order to remove secretion from the tympanum.

SUMMARY.

A number of past and present day views on effusion into the tympanum have been enumerated. Although known and carefully described nearly 100 years ago, attention has been

centered on this condition again in the past decade. This is in all probability due to the widespread use of antibiotics in upper respiratory infections, sterilizing the middle ear but leaving a noninflammatory secretion in the tympanum. In obstinate cases that do not respond to simple inflations, an incision low down anteriorly is strongly recommended, perhaps with a counter-opening posteriorly. This combination gives uniformly good results in the vast majority of cases.

In conclusion it cannot be emphasized too strongly that conservative treatment should be given a thorough trial before myringotomy is considered.

BIBLIOGRAPHY.

1. Personal communication from Erik Ruttin.

2. Politzer, Adam: Diseases of the Ear (1869). Revised by Milton J. Ballin. Ed. 6. Philadelphia, Pa.: Lea & Febiger, 1926.

3. Stevenson and Guthrie: A History of Otolaryngology. E. and S. Livingstone, Ltd., 1949, p. 71.

4. Eagle, W. W.: Secretory Otitis Media. *Ann. Otol., Rhinol. and Laryngol.*, 55:1:55, 1946.

5. Jordan, Ray: Chronic Secretory Otitis Media. The Laryngoscope, 59:1002-1015, Sept., 1949.

6. Lawson, Lawrence J.: Secretory Otitis Media; with Special Reference to the Mucus Type. *Arch. Otolaryngol.*, 6:346-352, Oct., 1927.

7. Lowy, K.: Concerning the Pathology of Secretory Otitis Media. *Monatsschr. f. Ohrenh.*, 72:40, 1938.

8. Robinson, J. Mathews: Subacute Catarrhal Otitis Media and Mastoiditis with Effusion. *South. Med. Jour.*, 35:815-825, Sept., 1942.

9. Senturia, Ben H.: Etiology, Pathology and Treatment of External Otitis. Instruction Course, American Academy of Ophthalmology and Otolaryngology, 1948.

10. Tobey, Harold G.: An Acute Catarrhal Otitis Media with Effusion. *Trans. Amer. Otol. Soc.*, 36:51-64, 1948.

11. Hoople, Gordon D.: Otitis Media with Effusion: A Challenge to Otolaryngology. *Trans. Amer. Acad. Ophth. and Otolaryngol.*, p. 531, 1950.

12. Hickey, Harold L.: Nasopharyngeal Malignant Tumor; an Overlooked Condition. *Arch. Otolaryngol.*, 53:1:58, Jan., 1951.

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SARCOMA OF THE NOSE AND SINUSES.*

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The American public has become cancer conscious. With the numerous articles appearing in current digests, newspapers, and women's magazines, the physician sometimes is hard put to keep ahead of his patients in the latest news on cancer detection and treatment. This is a desirable state of affairs, and now more than ever before, it behooves every specialist to have a wide and detailed knowledge of all malignancies that could occur in his field of practice.

Sarcoma arising in the nose or paranasal sinuses is a rare entity, but since two cases have occurred in our practice within a relatively short time, I considered the condition worth some special investigation, especially since both patients had been treated by other physicians who did not suspect malignancy.

History. Sarcoma of the nose and sinuses has been known and recognized for many years. Bosworth,[1] in 1889, published a book, "Diseases of the Nose and Throat," which included a chapter on sarcoma of the nasal passages. He collected 41 cases, about half of which were said to arise from the nasal septum. Most of these had not been examined microscopically.

The first published description of sarcoma of the nasal septum was a case report by Paletto, in 1820, and the first case which included an adequate microscopic study was reported by Mason, in 1875, according to R. H. Johnson, who

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reviewed the history of this lesion in 1904.[2] Clark (1898),[3] Baker (1899),[4] and Craig (1901)[5] also contributed early reports.

Etiology. The causes of sarcoma arising in the nose and sinuses are as obscure as those of most neoplasms elsewhere. In the literature no definite etiological factor appeared in most cases. In no case was trauma, chronic irritation or infection suspected as a probable cause. Irradiation, however, had a causative rôle in five instances.

Osteogenic sarcomas have been produced experimentally by means of X-rays and radium. Also cases of bone sarcoma have been reported after beta and gamma radiation. Wolfe and Platt[6] reported two cases of sarcoma occurring in the nasal bones in the region of the glabella. Both patients had received irradiation for squamous cell carcinoma of the skin in this area 15 and 20 years previously. Diagnosis of sarcoma was confirmed by microscopic sections of the tumor in each instance. They report a case published in the German literature by H. Hase, of a 12-year-old boy who developed sarcoma of the nasal bones. This child had had a scar over the nose, treated by X-ray irradiation at two years of age. They found 60 cases of widespread sarcomas reported in the literature up to 1948, where osseous sarcoma could probably be attributed to the effects of Roentgen ray and radium therapy.

W. G. Cahan[7] reported 11 cases of sarcoma developing after irradiation for some other cause. Two cases involved the sinuses. One was in a 26-year-old male who developed a sarcoma of the antrum 13 years after irradiation for a cloudy left antrum. Radical surgery was done and a three-year cure resulted. The second case was an osteogenic sarcoma of the ethmoid labyrinth which first appeared five years after irradiation for retinoblastoma in infancy. The tumor was radically excised; but it recurred, and the child died.

These cases should again remind us of the inherent dangers in radiation therapy. Its use must be reserved for potentially serious situations, and complications of treatment must be watched for constantly.

The so-called "cancer age" is not an important factor in sarcoma of the nose and sinuses. Many of the reported cases have been in children, and Pancoast[8] believes the disease to affect young people more often than the middle-aged; however, there has been only one case of sarcoma of the antrum diagnosed in approximately 90,000 admissions to Children's Hospital in Milwaukee in the last 26 years. This occurred in 1939 in a 10-year-old boy who died in spite of X-ray therapy. It is the impression of most authors that malignancy of the nose and sinuses in childhood is more rapidly growing and the prognosis is worse than when it occurs in later years.

Diagnosis. There are no typical symptoms of sarcoma, but certain findings may be suggestive. Some authors state that these tumors are slower growing with less tendency to ulceration than the carcinomas, and they present more distinct expansive growth with extension by contiguity, and symptoms due to pressure, rather than invasion and destruction as with the carcinomas. Nasal obstruction and pain are the two most frequent complaints. Wille,[9] reviewing 220 cases of malignancy at the Norwegian Radium Hospital, lists these presenting symptoms in order of frequency:

Nasal stenosis	41	Canthal swelling	8
Jaw and toothache	38	Anesthesia of N V	6
Pain in eye and temple	32	Loose teeth	4
Swelling of cheek	27	Impaired vision	3
Nasal discharge	21	Enlarged cervical glands	2
Alveolar tumor	12	Exophthalmos	2
Epistaxis	11		

X-ray examination is helpful in the diagnosis, and Pancoast[8] believes that sarcoma of the antrum can usually be diagnosed early by Roentgenograms. Homogeneous clouding of the antrum is present, but the appearance is not so dense as the clouded sinus due to polyps or thickened membranes with retained secretion. The original antral lesion is usually in the region of the teeth with the bone quite thin in that region. Alterations in alignment of the teeth due to encroachment by the lesion in the sinus is a later finding, and finally expansion and thinning of the walls of the sinus to a marked degree as the lesion increases in size. The bony outline of the

sinus may disappear altogether. Fibroma is the only tumor which simulates sarcoma. These Roentgen findings confirm the clinical impression that the sarcomas are expansive rather than invasive growths. The contrasting X-ray picture of carcinoma of the antrum is one of erostion and destruction of the bony walls. The bone has a speckled or mottled appearance as though it was the seat of bone disease or invasion by a new growth.

The chief Roentgen sign of sarcoma of the ethmoid area, too, is the expansion of involved structures rather than invasion. For this reason it is often difficult to differentiate primary sarcoma from mucocele.

Final diagnosis depends upon examination of biopsy material or upon frozen section examination at the time of operation.

Pathology. The pathology of sarcoma is difficult to describe because of the various elements which may go into its formation. The amount of fibrous, cartilaginous, myxomatous, and osseous tissue and degree of cellularity, all are variable. Ohngren,[10] reporting from Stockholm in 1933, found 22 sarcomas (or 15 per cent) among 129 cases of malignancies arising in the maxilloethmoidal region; these were of the following types:

Osteoblastic	5	Spindle cell	2
Round cell	3	Plasmocytoma	3
Polymorphous	3	Myxosarcoma	2
Melanosarcoma	3	Fibrosarcoma	1

Ringertz,[11] of Helsingfors, has done a complete study of pathological types of sarcoma. He found that 5 per cent of all nasal and paranasal sinus tumors were sarcomata; that of this number, 20 per cent were fibrosarcoma or spindle cell sarcoma. Next in frequency were the myxomas and myxochondrosarcomas, and lastly the osteogenic sarcomas. In his experience this last group was by far the most malignant.

Most pathologists agree that the diagnosis cannot always be made from the histological picture alone. The clinical behavior of the tumor must also be considered. A growth which

from histological appearance alone might be called a chondroma would have to be considered sarcomatous if it was locally recurrent and invasive. Such situations arise not infrequently. Also, in recurring, these tumors often become more cellular and assume a more malignant character.

Treatment. The evolution of treatment of malignancies of the antrum can be traced in a series of papers by G. B. New.[12-15] In reviewing the literature from 1926 he discovered that treatment by surgical excision had been generally unsuccessful and that irradiation alone had likewise produced poor results. In 1920,[12] New advocated eradication of the tumor by the actual cautery and implantation of radium into the marginal tissue. Later experiences led to the use of electrocautery instead of open cautery with radium. By 1926,[13] New had collected 97 cases of antral malignancy, 30 per cent of which were apparent cures. Half of these favorable cases were sarcomas. He reported further results in 1935[14] and 1938.[15] About 25 per cent of the total cases were sarcoma, and in these the results were particularly favorable — as high as 70 per cent five-year cures in one series.

It was pointed out by both Ohngren[10] and Holmgren,[10] who also favor this combined electrocautery-irradiation technique, that sealing of lymph and blood channels by the current will help prevent extension and recurrence. With ordinary surgical procedures there is more blood loss and more likelihood of spreading tumor cells to uncontaminated fields. Ohngren[16] reports 35 per cent five-year cures of maxillary sinus tumors but does not differentiate the types.

I. Szpunar,[17] of the Cracow University Clinics, uses electrosurgery as an adjunct to X-ray therapy rather than the reverse but emphasizes its importance in facilitating drainage, lessening infection, and promoting healing. He discusses 51 cases of malignant tumors of the maxillary sinus, of which five were sarcoma. He also mentions the use of nitrogen mustards in far advanced cases, feeling that they alleviate pain and temporarily improve the patient's general condition.

Each case must, of course, be individualized. If the sarcoma is early, readily accessible, and of low grade malignancy, electrosurgical excision is enough. Exact extent of the malignant tissue is, however, often difficult to determine; and if there is some doubt that excision is complete, prognosis can be improved if radium is implanted at the time of surgery.

If frozen section or biopsy shows a highly cellular tumor, some form of irradiation is mandatory in combination with electrosurgery.

When there is evidence of metastases, prognosis is grave and surgery useless, but X-ray irradiation will frequently reduce the size of the tumor and alleviate pain temporarily.

New[15] is of the opinion that the patient with a high grade malignancy has a better chance of primary cure by electrosurgery plus radium implants than one with a low grade tumor where radiosensitivity is low and surgery alone must be depended upon.

CASE REORTS.

Case 1: Mrs. A. G., age 40, was referred to us by an ophthalmologist in January, 1946. The patient had complained of a dull aching pain over the right frontal sinus and right eye, and a feeling of fullness in this region since October, 1945. Several severe, right-sided nose bleeds had also occurred. The doctor had recently operated for what he thought was a chronically infected lacrimal sac on the right, but he did not complete the operation because of hemorrhage. Positive physical findings were limited to the head. A hickory nut sized swelling was seen above and medial to the right inner canthus, and there was profuse tearing of the right eye. Vision was normal. The ears, throat, nasopharynx and larynx were normal. Intranasally there was some granulation-like tissue in the right middle meatus. This tissue bled easily on slight manipulation. No pus was seen in the nose, but the tissue present suggested some trouble in the sinuses on the right side.

X-ray changes were minimal considering the size of the external swelling. There was an asymmetry of the frontal sinuses particularly in the region of the frontoethmoid cells. There was lack of trabecular formation on the right side, but no actual bone destruction nor tumor formation could be seen (see Fig. 1, Case 1).

A tentative diagnosis of mucocele was made.

A right intranasal ethmoidectomy was attempted in April, 1946. Tissue was obtained for microscopic examination, but operation could not be completed because of hemorrhage. The pathologist reported inflammatory tissue. We planned to complete the operation through an external

approach, but the patient found it necessary to leave the hospital because of a family emergency. We were able to see her occasionally in the office, and each time she was urged to have the operative work completed. Her husband remained an invalid for many months, and the patient felt she could not re-enter the hospital. The external swelling increased slowly and the right eye began to protrude before we were able to hospitalize her again in February, 1948. The intranasal picture had also changed.

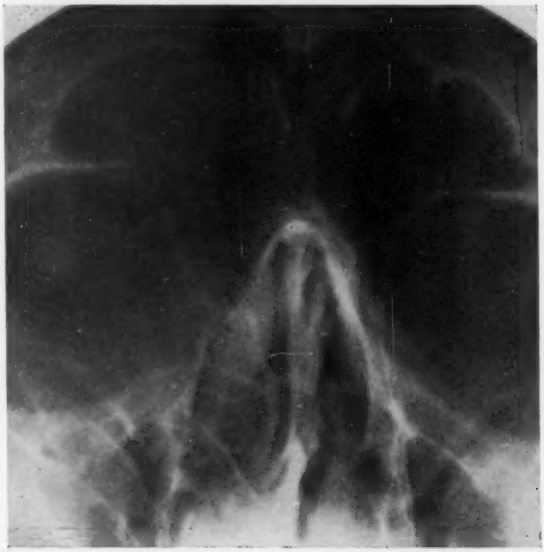

Fig. 1. Case 1. Waters projection taken February, 1946, showing some asymmetry of the frontal sinuses and lack of trabecular formation on the right, in the frontoethmoid region. No tumor demonstrated.

The right lateral wall of the nose was pushed against the septum, making it impossible to visualize the right middle meatus. X-rays at this time showed definite change. The anterior ethmoids, the anterior portion of the frontal bone in the region of the superior and inner aspect of the right orbit, and the septum between the two frontal sinuses could now no longer be demonstrated. There was a 1 cm. soft tissue shadow projecting from the right into the left frontal sinus (see Fig. 2, Caes 1).

Right external frontoethmoidectomy was done in February, 1948. Immediately beneath the skin and subcutaneous tissue, a very thin shell of bone presented itself. There were several areas where bone was absent, and sinus mucosa was seen. In removing bone for better exposure, we found what appeared to be a cyst wall. Practically all of the floor of the frontal sinus and the medial wall of the orbit were missing. The wall of the cyst varied from 1 to 3 or 4 mm. in thickness. A small amount of

thick mucus was found within the cyst. Bleeding was profuse throughout the operation, mostly from the eroded medial wall of the orbit. A 500 cc. whole blood transfusion was given during operation. A dehiscence was found in the septum between the frontal sinuses, and a bit of tissue protruded through the opening from the right into the left frontal. This tissue was removed, and the remainder of the frontal septum was excised. The mucosa of the left frontal was normal.

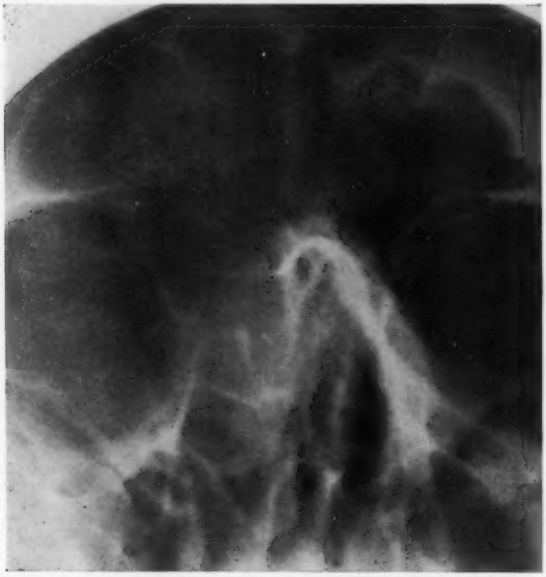

Fig. 2. Case 1. Granger 23° projection taken February, 1948. Here definite bone destruction in the region of the right anterior ethmoids and right orbit is seen. The bit of tissue, protruding from the right into the left frontal sinus found at operation, is clearly visible.

When operation was completed, all remnants of cyst wall, the entire floor of the right frontal sinus, the septum between the two frontals, and the lateral wall of the nose had all been removed. This left a large cavity which required tight vaseline gauze packing because of hemorrhage. The external incision was closed with black silk. Frozen section of the tissue was not done because of the benign appearance of the cyst and its contents. Fifty thousand units of aqueous penicillin were administered every three hours, and the patient was given three 500 cc. transfusions of whole blood on successive days because of continued bleeding. The postoperative course was otherwise uneventful. Packing was removed in stages over several days.

Microscopic examination revealed a cellular tumor, the cells of which closely resembled those of cartilage. The lesion was not encapsulated

and appeared locally invasive. Review of microscopic sections prepared in 1946, together with consideration of the course of the lesion, strongly supported the interpretation that this represented a neoplastic rather than an inflammatory lesion. Diagnosis: chondrosarcoma, low grade, ethmoid and frontal sinuses (see Fig. 3, Case 1). After learning the pathologic diagnosis, some form of irradiation was considered. The patient was three days postoperative at the time, and with the bleeding

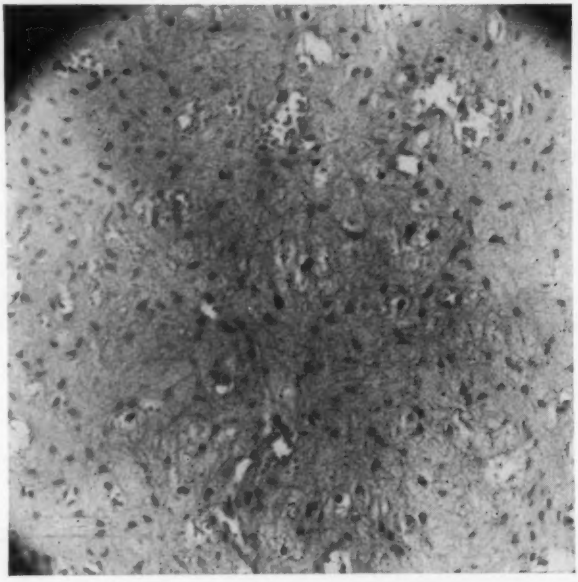

Fig. 3. Case 1. Photomicrograph of tissue removed at the time of operation, Feb. 25, 1948. Note irregularity of cells and lack of normal cartilage structure. (×100.)

the use of radium intranasally was not feasible. External irradiation might cause breakdown of the wound so that this method was rejected. From the pathological sections, we felt that this tumor was probably quite radioresistant and in view of the fact that we were reasonably sure all diseased tissue had been removed, we decided against irradiation.

The external wound healed nicely, and over a period of time the intranasal cavity epithelialized completely.

It was discovered on the third postoperative day that vision in the right eye was reduced to light perception only. An ophthalmologist felt that this was the result of our firm packing pressing either directly on the optic nerve, or on nutrient blood vessels to the eye—certainly an unexpected complication.

The patient has been observed in the office repeatedly. She was last seen in the office in March, 1950. There was no external swelling. Intranasally the operative cavity was completely clean and free of any recurrence. Vision remained poor in the right eye.

Comment. The loss of vision in one eye was a most unfortunate complication. Perhaps it could have been avoided by removing the packing sooner. A better method in theory would have been the use of electrocautery which might have made the tight packing unnecessary. Actually, we were following a line of cleavage in dissecting out the cyst and sharp dissection was necessary.

In retrospect, we think this to have been the course of the disease: A slowly developing mucocele of the right anterior ethmoids had most likely been present for several years without producing symptoms. In October, 1945, when epistaxis began, the sarcoma within the cystic mucocele probably had broken through into the nose. This occurred because the tumor was exposed in the middle meatus to any infection in the nose. External swelling was also observed at this time.

When the right external frontoethmoidectomy was done, the cyst wall was intact except in the middle meatus. This has led us to believe that this case represents the development of a sarcoma within a mucocele.

Case 2: We first saw the patient, E. V. P., age 50, July 25, 1947. She complained of nasal obstruction for which she had been under observation by an out-of-town doctor for one year. Local treatment by packing, the use of nose drops, and steam inhalations had failed to relieve her symptoms. An allergist, who had recently been consulted, felt that the obstruction was not on an allergic basis and referred the patient to us. There was no history of trauma or infection to account for the obstruction.

Ear, nose and throat examination was negative except for the nose. Externally the nose was normal. The nasal mucosa was pale, somewhat boggy and suggestive of allergy, and there was an excess of clear mucus in the anterior portion of both nasal chambers. Further back in the nose there was a symmetrical, smooth, bilateral swelling of the nasal septum, covered completely by intact mucosa. There were no areas of roughness, erosion or bleeding. The swelling began in the midportion of the septum, extending upward from the level of the superior border of the inferior turbinates, and backward to the posterior choanae. It was not possible to get an adequate view of either middle meatus or the middle turbinates. The inferior portion of the septum was uninvolved. On palpation the swelling was smooth and hard. The frontals were clear

and the antra hazy on transillumination. The nasopharynx was normal X-rays revealed a cystic swelling of the nasal septum without bone destruction. There was some clouding of the left antrum (see Fig. 4, Case 2).

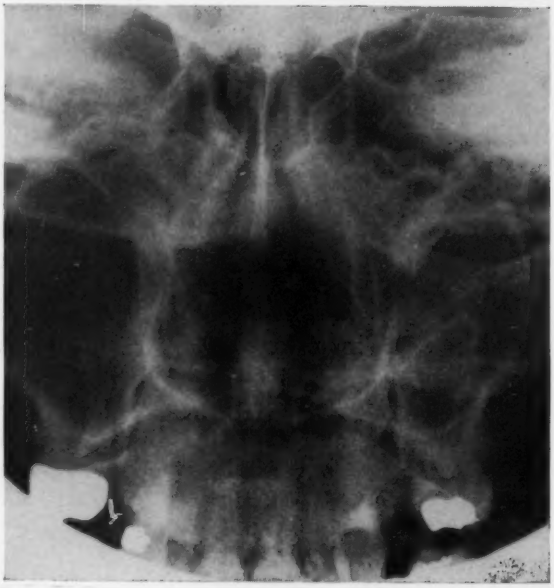

Fig. 4. Case 2. Granger 107° projection taken July, 1947, demonstrates the cystic swelling of the nasal septum. Some clouding of the left antrum is also noted.

Because of the length of time the symptoms had been present and its benign clinical and radiological appearance, our working diagnosis was a benign bone cyst of the septum, although we were unable to find any such condition described in otolaryngological or pathological texts. The possibility of a primary malignancy of the septum could not be excluded. Routine laboratory studies were normal and the patient had no fever.

It was decided to approach the lesion surgically by means of a standard submucous resection of the septum, and this was done on July 30. Anesthesia consisted of cocaine crystals moistened with 1/1,000 adrenalin applied topically. After separating the mucoperichondrial flaps in the usual manner and reaching the region of the swelling, a semisolid material was encountered. The perpendicular plate of the ethmoid was missing. The tissue was relatively avascular, but looked a great deal like malignant tissue, having the granular tapioca-like appearance of mixed salivary gland tumor or chondroma. Tissue was sent to the laboratory for frozen section. The pathologist's diagnosis was a highly cellular,

malignant growth; probably a sarcoma. All visible tumor was removed up to within 1 to 2 mm. of the cribriform plate. The tumor extended to the posterior choanae and had eroded, probably by pressure, the entire anterior wall of both sphenoids. It was necessary to curette laterally from the anterior wall toward the greater wing of the sphenoid on both sides in order to remove tumor tissue, but no tumor was seen within either sphenoid sinus. At completion of operation the mucosal flaps were still intact and normal on the external surface, but somewhat roughened on the medial side. All visible tumor had been removed.

Gauze packing was placed in the nose and left for 24 hours. Penicillin was given for one week. There were no postoperative complications.

Later, paraffin pathological sections (see Fig. 5, Case 2) were reported. Microscopic examination revealed the presence of a malignant tumor composed of cartilage, which in some areas was undergoing myxomatous

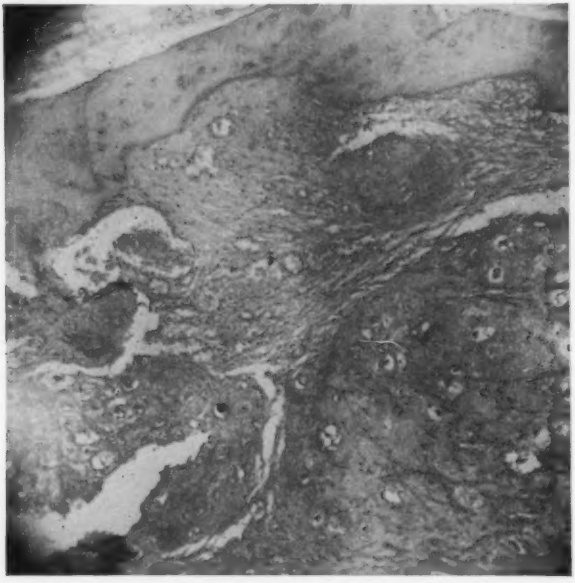

Fig. 5. Case 2. Photomicrograph showing section of tumor removed surgically, July 30, 1947. Atypical cartilage cells and myxomatous stroma are present. Also the tumor is seen invading and destroying bone and cartilage. (×100.)

degeneration. The tumor was invading and destroying the nasal septal cartilage and bone. Paraffin sections were not as highly cellular as the frozen section preparation, which would suggest that the tumor was not growing as rapidly as previously thought. The final diagnosis was chondrosarcoma of the nasal septum.

Since we could not be sure that our excision was adequate, we decided in consultation with the Roentgenologist to use some type of irradiation, in spite of the fact that this type of malignancy is reportedly not radiosensitive. On the ninth day after operation, 145 mg. of radium were put into the nose and packed as high as possible with vaseline impregnated gauze strips. It was left in place 12 hours, delivering 1,740 mg. hours. The only immediate postirradiation reaction was some dryness in the nose. Dryness and crusting of the nasopharynx, particularly on the left side, has been a problem. We have had the patient use plain olive oil in the nose to lubricate the nasopharynx, and she has also irrigated the nose with saline to help remove crusts. On three or four occasions there has been a little serous fluid in the left middle ear, which we have been able to relieve by inflations of the left Eustachian tube.

The patient has complained of occasional pain in the left side of the face and neck. No organic basis has been found to explain it, and we hope it is only a later effect of the irradiation therapy.

Ten months after irradiation had been given, a half dime sized ulceration was observed on the posterior pharyngeal wall. Biopsy of the margin of the ulcer showed only inflammatory tissue. It was decided that the ulcer was probably due to the radium, and it subsequently healed. The patient was last seen in August, 1950, at which time no recurrence of the tumor could be seen.

Comment. Primary chondroma of the nasal septum has been reported and is apparently not uncommon, but we have been unable to find a single case of chondrosarcoma similar to this case. Its site of origin in the septum cannot be questioned because we were fortunate enough to see the patient before it had spread to adjacent tissues. A cure cannot be claimed, but as no recurrence has occurred up to this time, we feel reasonably certain that none will.

SUMMARY AND CONCLUSIONS.

1. Sarcoma of the nose and sinuses is rare.

2. These tumors occur more often in childhood and adolescence than carcinomas. In most of the cases in the literature, no etiologic factors are evident, but in at least five cases sarcoma was preceded by irradiation to the region.

3. Sarcoma of the nose and sinuses grows by expansion rather than invasion, and has some characteristic Roentgen findings.

4. Exact pathological diagnosis is often difficult due to the various connective tissue elements which can comprise the tumor.

5. Treatment by electrosurgical excision augmented by radium therapy gives the best results in most cases.

6. Two cases are reported. The first case is unusual because a chondrosarcoma apparently developed within a mucocele. The second case is a primary chondrosarcoma of the nasal septum, a lesion which, so far as we know, has not previously been reported in medical literature.

BIBLIOGRAPHY.

1. Bosworth: Diseases of the Nose and Throat, 435. New York, N. Y.: William Wood & Co. Copyright 1889.

2. Johnson, R. H.: Sarcomata of the Nasal Septum. The Laryngoscope, 14:454, 1904.

3. Clark, J. P.: *Trans. Twentieth Annual Meeting, American Laryngological Association*, May, 1898.

4. Baker, H. R.: Adeno-Sarcoma of the Nasal Septum. The Laryngoscope, 7:213, 1899.

5. Craig, R. H.: *Montreal Med. Jour.*, 30:455, Jan., 1901.

6. Wolfe and Platt: Postirradiation Osteogenic Sarcoma of the Nasal Bone. *Cancer*, Vol. 2, No. 3, May, 1949.

7. Cahan, W. G., *et al.*: Sarcoma Arising in Irradiated Bone. *Cancer*, 1:3-29, May, 1948.

8. Pancoast, H. K.; Pendergrass, E. P., and Schaeffer, J. P.: The Head and Neck in Roentgen Diagnosis. Charles C. Thomas Co. Copyright 1940.

9. Wille, C.: Malignant Tumors in Nose and Accessory Sinuses. *Acta Oto-Laryngol.*, Supp. LXV, Oslo, 1947.

10. Ohngren, L. C.: Malignant Tumors of the Maxilloethmoidal Region. *Acta Oto-Laryngol.*, Supp. XIX, Stockholm, 1933.

11. Ringertz, N.: Pathology of Malignant Tumors Arising in the Nasal and Paranasal Cavities and Maxilla. *Acta Oto-Laryngol.*, Supp. XXVII, Helsingfors, 1938.

12. New, G. B.: Treatment of Malignant Tumors of the Antrum. *J. A. M. A.*, 74:1296, 1920.

13. New, G. B.: Malignant Tumors of the Antrum of Highmore; End-Results of Treatment. *Arch. Otolaryngol.*, 4:201, 1926.

14. New, G. B., and Cabot, C. M.: The Curability of Malignant Tumors of the Upper Arm and Antrum. *Surg., Gynec. and Obst.*, 60:971, 1935.

15. New, G. B.: Malignant Disease of the Paranasal Sinuses. *Amer. Jour. Surg.*, 42:170, 1938.

16. Ohngren, L. C.: Malignant Diseases of the Upper Jaw. *Jour. Laryngol. and Otol.*, 52:18, 1937.

17. Szpunar, J.: Treatment of Malignant Tumors of the Nasal Sinuses. *Acta Oto-Laryngol.*, 36:317, 1948.

ENDOTRACHEAL INHALATION ANESTHESIA.

SPECIAL REFERENCE TO POSTOPERATIVE REACTIONS AND SUGGESTIONS FOR THEIR ELIMINATION.*

PALUEL J. FLAGG, M.D. (by invitation),
New York, N. Y.

Last March I wrote each member of the American Laryngological, Rhinological and Otological Society, Inc., requesting his experience and judgment respecting the incidence of trauma in endotracheal intubation for anesthesia. In reply to the 514 inquiries directed to the total membership, including ophthalmologists, otologists and laryngologists, 154 responses were received. This total necessarily represents much more than the 30 per cent implied; in fact, it may be assumed that the majority of those interested in laryngology were kind enough to reply. These responses were assembled as a survey and a mimeographed copy was mailed to each respondee. This survey now appears in the April issue of the *Archives of Otolaryngology* released last week. My purpose today is to state more fully why this inquiry seemed necessary in the first place; second, to give you some of the reactions to it; third, to note a drift which is taking place in the whole field of anesthesia; and finally, to emphasize the details of the technique which, in my hands, has been almost entirely free from regrets.

May I first enlarge somewhat on the background upon which my use of this method was predicated.

It is common practice in medicine for a specialist in one field to turn to that of another when problems overlap. It is

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most unusual for men in one field to attempt to establish through experience findings referable to another; and yet, in the field of endotracheal anesthesia it would seem that the clinical reaction to instruments and technique has been employed to establish rules and regulations for present practice, rather than the experience of the laryngologist gained through an intimate acquaintance with physiological tissue reactions to instrumentation. How often, for example, does the laryngologist appear in the introduction of a new endotracheal tube or technique,—and yet, should not the use and safety of such instruments turn upon laryngoscopic experience rather than anesthetic trial and error?

Wherever endoscopy is used in other fields, the physician employing it lives with his patients through diagnosis, operation, immediate recovery and ultimate after effects.

In my initial use of endotracheal intubation, I turned to Chevalier Jackson. A carbon of a letter, dated Nov. 29, 1926, is before me. I asked Dr. Jackson the following questions: What is the usual reaction to intubation? What is the maximum outside diameter of the bronchoscope for the adult and the child? Would a flexible tube give less reaction? How long may the tube be left in the larynx without trauma? Will application of lubricants reduce the effect of intubation? Would a large size tube be less likely to traumatize than a smaller tube?

Jackson's replies were precise and afforded a safe base for action. Following further correspondence and trials, Jackson wrote me three months later (under date of Feb. 23): "I would suggest that you make a preliminary publication at the earliest possible moment." I replied: "No preliminary observation of my own would have any value. This would be merely speculation. You alone can pass judgment on the soundness of this method. If you say that such and such a technique, or a tube of such a size is correct, that is the end of it. Later on, in a further report of the method, my opinion based upon case experience will be of value."

For 10 years I did endotracheal anesthesia with the tubes recommended by Jackson, under every environmental condi-

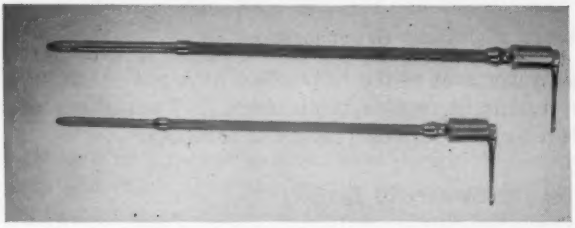
tion, in large hospitals, in small hospitals, in sanitariums and in homes, at leisure and under pressure. The results paralleled bronchoscopy. They were practically without regrets. New tubes, new laryngoscopes made their appearance; blind intranasal intubation became popular; carbon dioxide absorption technique, ethylene, cyclopropane followed each other. Through the daily contacts with which we are all familiar were voiced disturbing reports; seven granulomas in the practice of one laryngologist; sloughing of tracheal mucosa from balloon pressure followed by death; from another: stories of long struggles to intubate; an emergency call to assist in the intubation of a patient; apnoeic from oversedation; resistant to an attempt to intubate an oversized tube but offering no resistance or difficulty to the 10 mm. tube in our use; and then there was the threat behind it all: the threat that all endotracheal intubation might be condemned, including my own fortunate experience, unless the reasons for these accidents could be discovered. It seemed quite clear that the policy of trial and error to determine tolerance of the glottis, the attempt to accommodate the size of the endotracheal tube to the requirements of anesthesia machines was proving disastrous. A new and more complicated problem, one which I trust will not be judged presumptuous, was taking shape; *i.e.*, to investigate the rumors of trauma, to bring up to date the original postulates of Jackson, and to integrate these results with my personal experience for the benefit of the patient and for the protection of the field.

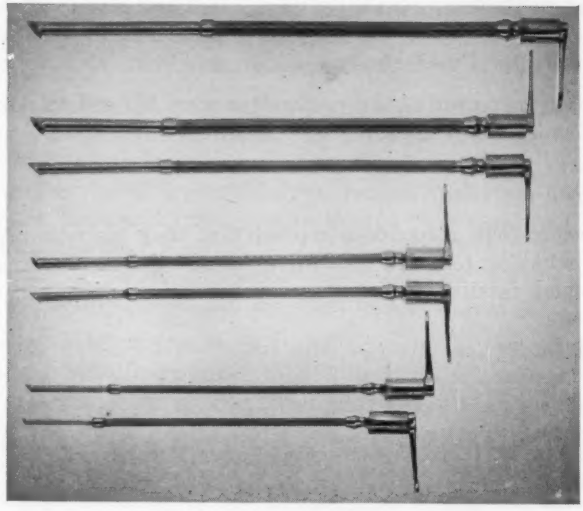
In 1926, I turned to Chevalier Jackson for advice. Through the last year I have come to you. You have responded most generously. You have reported pathology which has come to your attention following endotracheal anesthesia. You have confirmed the views Jackson stated to me 25 years ago.

May I now refer briefly to the questions placed, and to the *resulting summary* which appears in the April issue of the *Archives of Otolaryngology*.

Trauma Following Bronchoscopy.

In routine laryngoscopy and intubation for bronchoscopy 98 per cent expect little or no reaction.





Consultations for Trauma Following Anesthesia.

Ninety-nine respondees, or 64 per cent, were called in for postoperative trauma following endotracheal intubation for anesthesia.

Pathology Following Intubation for Anesthesia.

Thirty-four surgeons reported a total of 101 granulomas following intubation for anesthesia. Thirty-one reported a variety of lesions.

Pathology Following Bronchoscopy.

Eighty per cent of the respondees have not encountered the above lesions in routine bronchoscopy. Twenty per cent encountered minor lesions (except in infants).

Time of Appearance of Lesions.

Acute lesions appeared immediately or from two to 12 hours, chronic lesions—granulomas, appeared from four weeks to five months.

Necessity for Tracheotomy.

Thirty per cent of the respondees were obliged to do tracheotomies for trauma.

Deaths.

Twenty-four respondees replied that they were aware of deaths having followed intubation for anesthesia. Ten replies suggested fatalities.

Blind Intubation.

Ninety per cent condemned blind intubation.

Maximum O. D. in Endotracheal Tubes Recommended.

Ninety-five per cent restricted the infant tube to 4 mm. O. D. Ninety-two percent limited the adult O. D. tube to 10 mm.

What has been the reaction of individual members of the Triological to this survey, to which they have contributed? I shall note a few of these comments:

From Connecticut: Today most endotracheal intubations or anesthesia are done using pentothol as the basic anesthesia. Most anesthetists simply do not take time to anesthetize the throat and larynx properly with local anesthesia. When spasm occurs, as it frequently does, then trauma is produced in the hurry and excitement. The anesthetists know they will never have spasm to contend with if I fix the patient first.

THE EFFECT OF LONG INTUBATION IS REFERRED TO.

Louisiana: It is my opinion that bronchoscopy is less traumatic than endotracheal intubation for anesthesia due to the fact that bronchoscopy requires only a few minutes during which the bronchoscope is in contact with the vocal cords. On the other hand, the intubation tube frequently remains in contact with the vocal cords as long as several hours and this constant pressure, particularly if the patient is not under deep anesthesia, produces considerable trauma of the cords.

Massachusetts: I have often wondered if the prolonged presence of the tube in the larynx has any influence in the etiology of the traumatic granuloma which sometimes occur. On the other hand, I recall that prolonged intubation of the larynx for laryngeal diphtheria in the old days rarely caused any such trouble.

OBJECTIONS TO BLIND INTUBATION ARE REPEATED.

Pennsylvania: I have encountered a number of cases where the larynx was traumatized, and I have noted many more cases of traumatism on the nasal passages, due unquestionably to the blind intubation through the nose, of which I heartily and thoroughly disapprove.

Chicago: Certainly blind bouginage as well as the use of oversized tubes and intralaryngeal pressure are all to be condemned.

New York: I feel that this is a very important stand for you to take as I have seen a great many cases with hoarse voice, over months and months, and also cases where nose

bleed developed following intratracheal anesthesia. Most of the cases I have seen have been cases that were induced by the blind method of nasal catheter.

THE EFFECT OF LOCAL ANESTHESIA LUBRICANTS ON THE ENDOTUBE IS POINTED OUT.

California: I would like to add that the worst immediate reactions to intubation that I have seen were traceable to the use of anesthetic lubricants, nupercaine and intracaine, causing contact dermatitis, so that it is not surprising that an occasional patient will show a pseudomembranous reaction in the larynx and subglottic region. I have not seen this reaction to intubation except where such agents were used.

THE FOLLOWING COMMENTS STRESS ACCIDENTS.

New York: Let me mention one case which I blame in part on endotracheal anesthesia. This was in a boy who had been run over and had a ruptured spleen. There was no evidence of injury to his chest. In doing the spleenectomy, endotracheal anesthesia was employed. He had respiratory difficulty on the table which prevented removal of the tube after the operation was completed. Two hours later he was dead, and at autopsy it was discovered that he had a massive tension pneumothorax from a tiny laceration in the monor fissure of his right lung. I accepted most of the blame for this, because I should have recognized that there was a tension pneumothorax; but, on the other hand, if the anesthetist had not been pumping anesthetic gas into him by squeezing on the bag, the boy would not have had the tension.

I know of three deaths due to improper intubation and a fourth was prevented only by wide incisions in the neck the day after operation. Two of these were cases of my own.

New York: I had to stand by for hours while a case that was in great respiratory distress was being considered for a tracheotomy. The patient had endotracheal anesthesia for a thyroidectomy. Both cords were immobile and in the midline,

but the vocal processes moved on attempted phonation, which ruled out a paralysis of the recurrent laryngeal on both sides. The consensus of opinion in this case was that the larynx was damaged by trauma of endotracheal anesthesia.

I have attempted to investigate rumors of trauma following endotracheal anesthesia. The recommendations made to me by Jackson 25 years ago are almost identical with those which you have stated and which now appear in the April issue of the *Archives.*

Your responses indicate that laryngoscopy and intubation in bronchoscopy is relatively nontraumatic, that laryngoscopy and intubation for anesthesia is markedly so.

The first step to take in establishing the etiological factors responsible for this reaction would seem to be to note the variances in the methods.

The first difference is the intubation of an unconscious patient. The mechanical variances to be noted are larger tubes, blind intubation and the use of subglottic packs. If the reasons for the pathology reported are to be found in these variances, their removal should eliminate them.

While the survey offers no broad evidence to bear out the logic of this conclusion, it is a matter of revealing interest to reflect that by reason of conservatism or, possibly, lack of originality, the endotracheal tubes which I have used have never exceeded the outside diameter which you recommend. I do not use blind intubation. I do not employ subglottic packs; first, because I have found such tubes too bulky; and, second, I have found them unnecessary. My patients have always been adequately relaxed with ether. Postoperative trauma in my hands has not exceeded that which you report for routine bronchoscopy. As a matter of fact, it has been less than 2 per cent.

I now comment upon the matter of general anesthesia, the field obtainable under ether anesthesia, the instruments which I have used and, finally, what I believe to be the implications contained in the popularization of laryngoscopy and intubation in the relaxed patient.

May I point out what seems to me to be a major drift affecting the whole field of anesthesia? A realization of this drift will help to eliminate much of the mystery presently complicating anesthetic practice and postoperative reactions.

It seems clear that anesthesia has drifted from pneumatology, the use of gases, to pharmacology, the use of drugs, administered by mouth, rectum, hypodermic, vein and spinal canal. This drift is accompanied by the complications common to pharmacology. Dosages are irreversible, effects are often unpredictable and idiosyncrasies are not uncommon. In therapeutics, pharmacology anticipates these complications and guards administration by establishing a therapeutic index for each drug, a margin of safety between the effective medical dose and the lethal dose.

When, however, drugs are called upon to eliminate consciousness, muscle tone and reflex activity, to depress respiration to the vanishing point, what has become of the therapeutic index, the margin of safety? Furthermore, when one depressant drug is added to another, in order to produce a suitable surgical field, is not the result shrouded in mystery?

What is back of this drift? Is it not ease of administration, an agreeable induction and euphoria upon recovery skillfully integrated and attractively presented by pharmaceutical firms? The power of these motivating forces are great. An appeal to safety and controllability may induce the patient to tolerate gas therapy; it will not make it agreeable. The foregoing is not intended as a blanket condemnation of the substitution of pharmacology for pneumatology. It is an attempt to bring into perspective the limitations as well as the usefulness of these sciences to help the surgeon to anticipate the normal limits of control of gases and drugs. In the light of these facts, one may more easily appraise and assist present practice in anesthesia.

While pharmacological control, with preoperative sedation, morphine, scopalamine, pantopon or demoral, with loss of consciousness by intravenous pentothal-sodium, an agreeable

induction is begun. This medication does not accomplish muscle relaxation or reflex control of the glottis. The field at this point resembles that of laryngoscopy and intubation under local anesthesia. To complete the preparation of the field by the pharmacological approach, curare or syncurine is used for relaxation, and topical pontocaine or cocaine is used for control of the glottic reflexes. The doses of these drugs and the individual's reaction vary. Increased control is secured at the cost of reducing the margin of safety. Pharmacological anesthesia is common practice. The skill required for its use is largely that of venapuncture and observation of the effects of the drugs used. Antidotes are singularly ineffective in unexpected reactions. Emergency measures for relief of laryngospasm and respiratory depression turn upon positive pressure, insufflation, through the endotracheal tube.

Anesthesia is maintained by virtue of the continued effect of preoperative sedation to which is added barbiturate hypnosis. Recovery is likely to be delayed and is occasionally complicated by the characteristic barbiturate respiratory depression which cannot be overcome by the use of carbon dioxide. As a rule, the patient awakens free from unpleasant effects and with an inclination to euphoria.

Pneumatological control of general anesthesia, carried out with gas-oxygen-ether, is, on the other hand, reversible in its effects; its effects are predictable to a high degree but prefaced by an induction which is sometimes unpleasant.

Maintenance is under an elastic control which leaves a wide margin of safety between adequate muscle relaxation, control of the glottic reflex and depression of the respiration. Where such anesthesia is induced without sedation, as is my practice, induction is more rapid, relaxation is more easily secured and recovery prompt. Patients recovering from ether anesthesia with endotracheal control, as a rule, have little nausea or vomiting. This is particularly true in operations about the head and neck. A brief period of recovery is followed by a prompt return of normal vigor. Where two or three children are anesthetized one after the other for squint operations, the

first is usually sitting up in his crib when the third is returned to bed. With the exception of occasional preliminary avertin, the speaker prefers to use no preliminary medication. Ether usually provides a relaxed field in from eight to 12 minutes.

A word as to this relaxed field is in order. There is a sharp and most important difference between laryngoscopy and intubation of the conscious patient under local anesthesia and the patient who is limp from adequate general anesthesia or who is in the flaccid stage of asphyxia. In the first, there is the familiar resistance of muscle tone and a variable degree of reflex activity. In the second, exposure is no more difficult than is exposure by a tongue depressor or nasal or oral speculum. This difference is so real, and its importance in instrumentation is so great that the laryngologist who limits his work to exposure under local anesthesia should go out of his way to familiarize himself with the freedom of exposure in relaxation. A field which is suitably relaxed brings this instrumentation within the range of many who would otherwise be excluded as unskillful.

Given a relaxed field, in which the glottis may be exposed and intubated with ease and without haste, much of the objection which has been raised to the wide use of laryngoscopy from the trauma caused by tense muscle and reflex spasm, would disappear.

Would it not be well worthwhile for the laryngologist who is faced with anesthetic difficulties to laryngoscope and intubate his own patient, thereby eliminating the chief hazard to head and neck anesthesia?

Now a word regarding instruments: it will be found that a laryngoscope blade at least seven inches long, with a large light and an opening to the side will provide a better field of exposure and allow easier intubation than does the conventional laryngoscope.

The tube which I have used has been previously described in detail. (Reference will be found in the April issue of the *Archives*). A set of seven tubes are now available, beginning

with 4 O. D. up to and including 10 mm. O. D. While some prefer to connect the endotracheal tube to a carbon dioxide absorption canister or to a rebreathing bag, I have found that what has been referred to as the "tin can" inhaler leaves little to be desired. Inspiration and expiration are entirely free. Negative and positive pressure, 2 or 3 mm., in the closed inhaler may not seriously inconvenience the patient, but complete freedom is preferable.

The purpose of endotracheal inhalation anesthesia is, first, to provide unobstructed respiration, in adequate volume; second, to prevent aspiration of foreign matter from the pharynx. Beecher has shown that the tubes which I have employed provide adequate ventilation (Henry K. Beecher, *Anesthesiology,* Vol. II, No. 6, 1950). Large tubes and tubes with subglottic balloons have been employed by many to reduce aspiration around the tube. I have found that a pharyngeal pack introduced over the top of the tongue deep into the pharynx prevents regurgitation of gastric contents into the hypopharynx and the draining into it of secretions from the mouth. The occasional seepage of fluid past the tube is immediately evident in the sound of the respirations through the tube. Such secretions are immediately removed by suction catheter.

One of the most important factors of safety during both induction and maintenance of endotracheal anesthesia is adequate suction. Its presence in the anesthetizing and operating room is mandatory.

At this point I should like to submit for your consideration a new tube which illustrates the principle of the Mosher lifesaver. A 5 and a 7 mm. endotracheal tube terminates in a tapered catheter-like tip. This tip is supplied with perforations, the total area of which is more than twice the area of the lumen of the tube. This new tube has a double purpose: first, it provides a means of entering a spastic glottis without trauma; second, it serves as a practice instrument for the student learning to intubate.

The need for this tube has been precipitated by the frequency and the danger of severe laryngospasm accompanied by respiratory depression reported during pentothal anesthesia. The ease and the frictionless entrance of this lubricated tube through a spastic glottis must be experienced by the operator in order to be appreciated. It is believed that this type of tube will serve a useful purpose in accidental asphyxia requiring intubation.

In conclusion, may I refer to the need of popularizing laryngoscopy and intubation in the relaxed, asphyxiated patient? Where one is about to die from respiratory obstruction and where the only possible relief is in the hands of rescue squads, it is becoming increasingly evident that at least one member of each squad should be oriented in the death zone of the respiratory tract, taught how to relieve pharyngeal obstruction and instructed in the simple technique of intubation of the completely limp patient.

To this end I have for four years given regular monthly courses of laryngoscopy and intubation for the treatment of acute asphyxial accidents, to pediatricians, obstetricians, hospital residents, nurse-technicians, and selected members of rescue squads.

Orientation of the field has been accomplished by the use of anesthetized dogs, anesthetized patients, and cadaver. The saving of many lives has resulted.

This course is given monthly and is completed in a Friday afternoon and Saturday morning session. The fifty-first class met last Friday and Saturday.

SUMMARY.

The importance of cooperation between the fields of laryngology and anesthesia is stressed. Pathology following endotracheal anesthesia is discussed. The present drift of anesthesia from pneumatology, the use of gases, to pharmacology, the use of drugs, with its implications, is stressed. The difference between laryngoscopy in the conscious patient un-

der local anesthesia and in the unconscious patient, relaxed by general anesthesia or asphyxia is noted. Instruments used by the writer are referred to, and a new lifesaver tube is demonstrated. A plea is made for the popularization of laryngoscopy and intubation in the relaxed patient with an obstructed airway. Courses of instruction for all those responsible for the care of the unconscious patient are offered.

FRONTAL SINUS: PERMANENT DRAINAGE WITHOUT DEFORMITY.*

HARRY P. SCHENCK, M.D.,
Philadelphia, Pa.

While surgical approach to the frontal sinus may become necessary in the management of neoplasms, the removal of foreign bodies, or the reduction of fractures of the frontal bone, it is usually carried out because of the spread of infection to the bony walls or beyond the confines of the sinus.

Since the introduction of sulfonamides and antibiotic drugs, conservative measures, together with chemotherapy, have controlled most acute infections of the frontal sinus, reducing the incidence of complications and of chronicity of infection.

In spite of the advances in chemotherapy, there are occasional instances when extension of infection takes place to the bony walls, subperiosteal abscesses or draining, fistulous tracts appear, and meningitis, osteomyelitis or brain abscess become complicating factors. Neglect, delayed or inadequate therapy, the use of the wrong chemotherapeutic agent, inept intranasal intervention or the presence of structural defects probably account for most of these sequelae.

Chronic sinus infections are resistant to antibiotic therapy because of the difficulty with which the drugs penetrate abnormal tissues resulting from repeated or prolonged infection. In the case of the frontal sinus, once irreversible tissue changes occur within the sinus and the nasofrontal ostium reinfection and chronicity of infection are inevitable and complications common.

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Regardless of the reasons for external surgical approach to the frontal sinus, the selected technique should accomplish the aim of the operation with the least residual deformity and scarring. Since the introduction of antibiotics, these objectives are usually attained by minimal sacrifice of sinus walls, the construction of a permanent drainage tract from the frontal sinus to the nasal cavity and immediate closure of the external wound. During the preantibiotic era, when wide removal of all diseased bone was imperative because of the lack of known controls against the spread of infection within bony structures, the operations of Riedel, Killian, Lothrop and Lynch were lifesaving measures. Today they are often no more effective than simpler procedures and entail needless sacrifice of functioning structures and membranes.

Osteomyelitis, with or without brain abscess, has rarely been cured by antibiotic therapy alone, but antibiotic therapy together with surgical drainage has effected some remarkable recoveries. Even the view that all necrotic bone must be removed before healing can occur may need revision. Although the antibiotics exhibit a peculiar ability to relieve the pain associated with osteomyelitis, their main value lies in the prevention of sequestration and in the arrest of progressive extension to contiguous osseous tissue. Their timely use in osteomyelitis secondary to sinus infections has not reduced the need for surgical drainage but it has reduced the extent of radical surgery. The question today is, "What constitutes adequate surgical drainage?"

After external entrance to the frontal sinus and the completion of the necessary intervention within it, an efficient drainage tract from the sinus to the nasal cavity must be maintained. Following either radical, deforming operations or more simple procedures, such a drainage tract is prepared by enlarging or reconstructing the nasofrontal approach. Because the subsequent constriction of the new channel is a serious matter, various methods have been employed to prevent it, either by the use of skin grafts in the frontal ostium and the nasofrontal channels, or by the introduction of tubes of tantalum, plastic or rubber.

The following simple technique has been employed for many years in the absence of bone infection, but since the introduction of the antibiotics, it has been used to the exclusion of more radical operations excepting under unusual circumstances.

TECHNIQUE.

Twelve hours prior to operation, the site of the incision is outlined by a linear application of 25 per cent silver nitrate solution which follows the midline of the eyebrow. The hair-bearing surface is later shaved to insure surgical asepsis, and the predictions of subsequent objectionable effects of shaving have not been borne out in more than 100 operations.

Endotracheal anesthesia is preferable, but in the absence of serious infection, the operation can be readily carried out with local anesthesia. The incision through the skin is continued through the periosteum to the bone and its length restricted to the exact requirements of the operation. Digital pressure by an assistant controls the profuse hemorrhage from the branches of the supraorbital and frontal arteries and veins until all the bleeding points are secured. The periosteum is then elevated, most of the separation being confined to the lower flap. If the anterior wall of the frontal sinus is found to be intact it is entered just below the inner extremity of the eyebrow with a mastoid gouge, a motor-driven burr or a mallet and chisel. The opening is enlarged with a Kerrison bone forceps but its extent confined to the minimal requirements of the specific situation.

After attending to the lesions within the frontal sinus, a probe is introduced from the frontal sinus into the ostium in order to locate its position. Antrum rasps then are passed downward from the sinus through the ostium and to the nasal cavity. When the passage has been enlarged to accommodate a tube of 10 mm. diameter, a malleable probe, with an eye in its olive tip, is threaded with heavy silk ligature and passed from the sinus through the enlarged ostium to the nasal floor where the ligature is grasped with a hemostat and the probe

withdrawn, thus carrying the two free ends of the ligature to the frontal sinus. A rubber tube, long enough to reach from the lateral margin of the sinus to the floor of the nose is then attached to the nasal end of the ligature and its upper end drawn into the frontal sinus where it is guided in a gentle curve to the lateral extension of the sinus. That portion of the tube lying in the frontal sinus is fenestrated. It is often advantageous to remove the anterior portion of the middle turbinate to facilitate these manipulations. The profuse hemorrhage which occurs during the rasping of the passage subsides promptly as soon as the rubber tube is in position. The lower end of the tube is then cut off so that it terminates in the nasal vestibule. After careful approximation of the periosteal edges, the skin margins are approximated with No. 14 metal clips. The conspicuous ridge produced by the clips prevents separation of the skin margins during postoperative edema and minimizes subsequent scarring. After operation, the frontal sinus can be readily irrigated at any time by passing a No. 8 or 10 French soft rubber catheter through the drainage tube and lavage carried out with normal saline solution or azochloramide solution. The return of the irrigating fluids is thus free and no pressure is produced within the sinus. The drainage tube can be left in place for long intervals without discomfort, but usually epithelialization of the new tract is complete in six weeks or less. Removal of tube is accomplished by grasping the end in the nasal vestibule and pulling it out. No resistance is noted and there is no pain or discomfort during its removal.

A case history is presented to illustrate the results of this simple operative approach to the frontal sinus in the presence of several complicating factors.

REPORT OF A CASE.

A. R., a white woman aged 50 years, was admitted to the University Hospital on Dec. 9, 1949. She had had severe, boring pain in the left frontal region for five days, and there was edema and tenderness over the left frontal region, left cheek and left orbital rims. Admission was precipitated by the appearance of stiffness of the neck suggesting early meningitis.

She stated that she had been relatively well until October, 1948, when she developed severe left frontal pain, followed by swelling and tenderness over the left frontal area and edema of the left upper lid. Following the administration of penicillin and catheterization and irrigation of the left frontal sinus, she became symptom-free. In April, 1949, a similar attack occurred, but she again recovered after further catheterization and irrigation of the sinus and antibiotic therapy. Excruciating pain in the left frontal region again appeared seven weeks prior to the admission to this hospital. For five days the temperature ranged from 101.2° to 104° F. but returned to normal after intensive antibiotic therapy. At this time Roentgenograms revealed the presence of an osteoma in the posterior wall of the left frontal sinus.

On admission the left frontal area was not only swollen and injected, but a doughy character of the soft tissues could be palpated well beyond the hair line. The swollen lids completely closed the left eye, but there were no restrictions on extraocular movements. The left nostril was obstructed, and profuse purulent secretion was present in the left anterior middle meatus. Palpation also detected fluctuation and an apparent depression in the region of the frontal bone, and pus was aspirated with a needle. The spinal fluid was normal; there were 12 per cent neutrophiles and 23 per cent lymphocytes present in the blood with a W. B. C. of 12,600. During the first 24 hours after admission the temperature varied from 100.4° to 102° F., but remained normal thereafter.

At this time Roentgenograms revealed bone absorption in the left frontal region and extending as far posterior as the coronal suture. The report also pointed out that with chemotherapy manifestations of osteomyelitis of the skull have changed so that slight deossification is now the usual finding rather than the true "moth-eaten" appearance described in the past. The right frontal sinus was normal; the left frontal sinus was indistinct in outline with osteomyelitis alterations about its periphery. Dense clouding of the ethmoid cells, particularly on the left, was noted, as well as marked mucous membrane thickening in the right maxillary sinus and slight mucous membrane thickening in the left maxillary sinus. The sphenoidal sinuses were well developed with bilateral mucous membrane thickening.

Four hours after admission, the left frontal sinus was entered and thick, yellow pus escaped under considerable pressure. The anterior wall of the sinus was soft at the site of entrance and the mucosa within the sinus was so edematous as to fill the space. An osteoma 8 x 10 x 2 mm. was located in the posterior wall of the sinus and removed, exposing normal dura beneath. Temporary drainage was secured with the insertion of a rubber tube and the wound closed. The sinus was irrigated with azochloramide solution every six hours; 300,000 units of crystacillin were given intramuscularly every 24 hours and 500 mgm. of aureomycin were given every three hours by mouth. The patient remained afebrile.

Seventeen days after the first operation, the frontal sinus was reentered, the nasofrontal passage enlarged and a rubber tube introduced so as to reach from the lateral margin of the sinus to the nasal vestibule. The external wound was closed with metal clips. Two weeks later, a red, tender, fluctuant swelling appeared over the left frontal area near the hair line. When the pus aspirated from this abscess was cultured no growth was obtained. After further aspiration on two successive days, the swelling disappeared and the pain subsided. At this time the Roentgenograms showed still further destruction in the left frontal bone and a sequestrum appeared to be forming.

The patient remained free of symptoms and the drainage tube was removed at the end of six weeks. During the next three months antibiotic therapy was continued. Three hundred thousand units of crystacillin were administered every 12 hours and 50,000 units of penicillin every three hours intramuscularly, for six weeks after the final operation. During the following six weeks the patient received 500 mgm. of aureomycin by mouth every three hours and 300,000 units of crystacillin intramuscularly daily. All therapy was discontinued thereafter.

By Feb. 7, 1950, the zone of osteomyelitis could no longer be clearly delineated in the Roentgenograms, and on March 3, 1950, no reactivation of the inflammatory process could be observed in the films. Studies on May 5, 1950, revealed the bone defect in the left frontal region to be smaller; however, there continued to be a radiolucent area above the operative site, and although it appeared smaller than at previous examinations it seemed still to contain a small sequestrum which had decreased in size. In November, 1950, further X-ray studies revealed diminished size of the defect in the left frontal region, and the small sequestrum could not be identified. Since Dec. 6, 1950, Roentgenograms have failed to reveal a radiolucent area in the left frontal bone. The patient has been symptom-free for 15 months; the drainage tract from the left frontal sinus has not constricted and no abnormal secretions have drained from it.

The simplest external approach to the frontal sinus is adequate in the management of mucoceles, pyoceles and cysts of the frontal sinus and useful in the removal of foreign bodies and small osteomas. In selected cases it may also suffice in the control of osteomyelitis of the frontal bone.

Successful end-results depend upon permanent, continuous drainage of the frontal sinus, supplemented with the appropriate antibiotic agent in adequate dosage and administered over a long period of time or at least until Roentgenologic evidence of infection disappears.

The permanent drainage tract from the frontal sinus to the nasal cavity should be slightly more than 10 mm. in diameter. A rubber tube of 8 to 10 mm. diameter, if left in position within the tract for six weeks, will permit the regenerative ability of the epithelial cells of the nasal and sinus cavities to cover the surfaces of the new channel within a period of six weeks. Practically no deformity results.

LETTER TO THE EDITOR

C.I.D. AUDITORY TESTS W-1 and W-2.*

Central Institute for the Deaf, with the collaboration of Technisonic Studios, has prepared two new recorded spondaic word tests that resemble the familiar Auditory Tests No. 9 and No. 14 of the Psycho-Acoustic Laboratory (Harvard). These recordings are intended for use with speech audiometers for determining the threshold for speech. The new tests differ from PAL Tests Nos. 9 and 14 in that the vocabulary is confined to very familiar words, suitable for children as well as adults, and also the pace is faster and more suitable for talk-back instead of write-down responses.

The test material in both W-1† and W-2 is a list of 36 spondaic words (like *baseball* and *railroad*) which were selected from the larger group used in Psycho-Acoustic Laboratory Auditory Tests No. 9 and No. 14. The particular words were chosen because they are familiar and because these particular recorded items proved experimentally to be equally intelligible. Six different scramblings of the same list of 36 words have been recorded on discs. The items in each scrambling are identical transcriptions from the same original. The scrambling was done by cutting and splicing the magnetic tape on which the transcriptions of the original were recorded.

In Auditory Test W-1 all of the words are recorded at a constant intensity and they monitor at approximately the same place on a VU meter. Each test word is introduced by the carrier phrase "Say the word." The carrier phrase, and also the 1000 cps calibration tone on the inner band of every record, have been recorded approximately 10 db above the

*This work was initiated under Contract N6onr-272 (Project No. NR142-170, T.O. III) with the Office of Naval Research and completed under Contract V1001M-577 with the Veterans Administration.

†Means Word Test Number 1.

average level of the test words. Auditory Test W-1 is designed for use with a step-attenuator to obtain the greatest possible flexibility and speed in finding thresholds for speech.

Auditory Test W-2 is a "descending level" test. The words in each of the lists are already attenuated on the record. After the first nine words the carrier phrases also start descending but they stay 6 db above the test words. The first group of words starts at the level of the calibration tone. Three words are at this level, the next three are down 3 db, the following three are down 6 db, and so on. Thus an average of 1 db attenuation per test word has been put into each list and each word that a listener repeats correctly lowers his threshold score by 1 db.

A subject's hearing loss for speech is defined as the number of decibels by which his threshold for speech is higher than the experimentally determined average threshold for a number of individuals whose hearing for pure tones in the speech range is within ± 5 db of the accepted norms for pure-tone audiometry. The value of the normal threshold for speech will vary not only with the particular sample of speech that is selected as test material but also with the equivalent that is employed. Neither the apparatus for speech audiometry nor the recordings of test material have yet been standardized, either by the Council on Physical Medicine and Rehabilitation of the American Medical Association or by the American Standards Association. It is, therefore, essential for each operator to establish his own normal values for each type of test with his own equipment and under his own particular acoustic conditions; however, if the recommendations concerning apparatus that are given in the manual that accompanies the new recordings are followed, normal threshold values obtained at other clinics should not differ from our own by more than 2 or 3 db. Our tentative values for the normal thresholds of speech are 20 db re 0.0002 dyne per cm^2 for Test W-1 and 19 db re 0.0002 dyne per cm^2 for Test W-2. A more extensive study is in progress to determine whether the small difference between the two figures represents an advantage for the pro-

gressive attenuation employed in Test W-2 or whether it represents a difference in the two groups of individuals employed for the two tests.

According to our preliminary measurements a speech audiometer that is calibrated for hearing loss should give a sound level of 20 db re 0.0002 dyne per cm^2 (measured in a National Bureau of Standards 9A coupler) when the calibration tone is set to the reference level on the meter and with the hearing-loss dial at zero. The starting level indicated on the hearing-loss dial minus the number of words of Test W-2 correctly repeated will then give the listener's hearing loss for speech directly within an accuracy of ± 2 db. A tone of 1000 cps at 20 db above 0.0002 dyne per cm^2 (under a receiver on the ear or on a 9-A coupler) corresponds approximately to the 1000 cps tone delivered by a properly calibrated pure-tone audiometer at the 5 db hearing-loss setting. This relationship is reasonable because even the most intelligible words must be a few decibels above the threshold of detectability before they can be understood.

The Technisonic Studios made the original recordings of the test material and also have carried through the entire process of manufacture including the electroplating and pressing. The Technisonic Studios will also distribute the recordings, on a non-profit basis, at the following prices:

W-2 as a set. 3 double-faced 78 rpm records, each containing 2 lists of W-1 @ $10.50

W-2 as a set. 3 double-faced 78 rpm records, each containing 2 lists of W-2 @ $10.50

W-1 and W-2 as a set. 3 double-faced 33⅓ rpm records @ $10.50

Individual records from the 33⅓ rpm set may also be purchased, as follows:

1 double-faced 33⅓ rpm record containing 4 lists of W-1 @ ..... $ 4.00

1 double-faced 33⅓ rpm record containing 4 lists of W-2 @ ..... $ 4.00

1 double-faced 33⅓ rpm record containing 2 lists of W-1 on one face, 2 lists of W-2 on the other face @ ..... $ 4.00

All recordings are on 12-inch discs.

The address of the Technisonic Studios is 1201 South Brentwood Boulevard, Richmond Heights 17, Missouri.

We hope that other laboratories and clinics that are interested in the standardization of speech audiometry will try these new recordings and communicate to us their criticisms and suggestions, and also any data that they obtain concerning the normal values for the thresholds of speech.

Signed: R. W. BENSON
H. DAVIS
C. E. HARRISON
I. J. HIRSØH
E. G. REYNOLDS
S. R. SILVERMAN

ILEME CONGRES FRANCAIS D'OTO-RHINO-LARYNGOLOGIE.

The Congrès Francais d'Oto-Rhino-Laryngologie will be held in the Grand Amphithéâtre of the Faculté of Médicine, Paris, Oct. 22, 23, 24 and 25, 1951, under the direction of the presiding Dr. M. Bouchet, of Paris, and Honorary President de M. le Prof. Léon Binet, member of the Institute.

For further information, address Dr. H. Flurin, Secrétary Général — 19 Avenue MacMahon, 17e, or Dr. H. Guillon, Assistant Sécretary Général, 6 Avenue MacMahon, Paris, 17e.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 1951-1952 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

July 1, 1951.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.
Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H Maico; Maico Model J.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model 45.
Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).
Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.
Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.
(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model J-92.

Manufacturer: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.
Distributor: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Vice-President: Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Meeting: Royal York Hotel, Toronto, Canada, May 18-19, 1952.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Royal York Hotel, Toronto, Canada, May 20-21, 1952.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President Dr. C. Stewart Nash, 108 Medical Arts Bldg., Rochester, N. Y.
Meeting: Royal York Hotel, Toronto, Canada, May 22-24, 1952.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich
Vice-Chairman: Dr. J. M. Robison, 1304 Walker Ave., Houston 2, Tex.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Royal York Hotel, Toronto, Canada, May 13-16, 1952.
Palmer House, Chicago, Ill., Oct. 9-12, 1951.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
President-Elect: Dr. Derrick Vail, Chicago, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 14-20, 1951.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. William B. Clark, 211 Loyola Ave., New Orleans, La.
Vice-President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

Meeting: Palmer House, Chicago, Ill., Saturday, Oct. 13, 1951.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Wm. H. Evans, 24 Wick Ave., Youngstown, Ohio.

President-Elect: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22, Pa.

Meeting: Chicago, Ill., Oct. 19, 1951.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

Meeting: Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.

Time and Place: Havana, Cuba, January, 1952. Dr. Jose Gros. For information write Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

SECOND LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

Time and Place: Sao Paulo, Brazil, July, 1951.

President: Professor A. dePaula Santos.

Secretaries: Dr. Jose de Rezende Barbosa, Dr. Plinio de Mattos Barretto, Hospital das Clinicas, Sao Paulo, Brazil.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Herman J. Moersch.

Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

Meeting: Royal York Hotel, Toronto, Canada, May 22-24, 1952.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Alden H. Miller.

Secretary-Treasurer: Dr. Victor Goodhill.

Chairman of Section on Ophthalmology: Dr. Dennis V. Smith.

Secretary of Section on Ophthalmology: Dr. Carroll McCoy.

Chairman of Section on Otolaryngology: Dr. Howard P. House.

Secretary of Section on Otolaryngology: Dr. Edwin Scobee.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.

Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.

Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21, N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. G. M. Billings, Morganton, N. C.

Secretary and Treasurer: Dr. MacLean B. Leath, High Point, N. C.

Time and Place: Sept. 11-13, Hendersonville, N. C. Jointly with the South Carolina Society of Ophthalmology and Otolaryngology.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Lewis F. Morrison, 490 Post St., San Francisco, Calif.
Secretary-Treasurer: Dr. Howard P. House, 1136 W. Sixth St., Los Angeles 17, Calif.
Meeting: Salt Lake City, Utah, 1952.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
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Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Courses annually the last two weeks in January at Los Angeles, Calif.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

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SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Francis LeJeune, Ochsner Clinic, New Orleans, La.
Vice-Chairman: Dr. V. R. Hurst, 315 N. Center St., Longview, Tex.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Dallas, Tex., Nov. 5-8, 1951.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
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SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Victor M. Noubleau.
Secretary: Dr. Héctor R. Silva.
1o. Vocal: Dr. Salvador Mixco Pinto.
2o. Vocal: Dr. Daniel Alfredo Alfaro.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

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Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
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Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
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Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

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Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Sec. de Actas: Dr. Juan Berini.

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Vicepresidente: Dr. César Cabrera Calderin.
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Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

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President: Dr. Joao Penido Burnier.
First Secretary: Dr. Gabriel Porto.
Second Secretary: Dr. Roberto Barbosa.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. F. J. Monteiro Sales and Dr. Jose Martins Rocha.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

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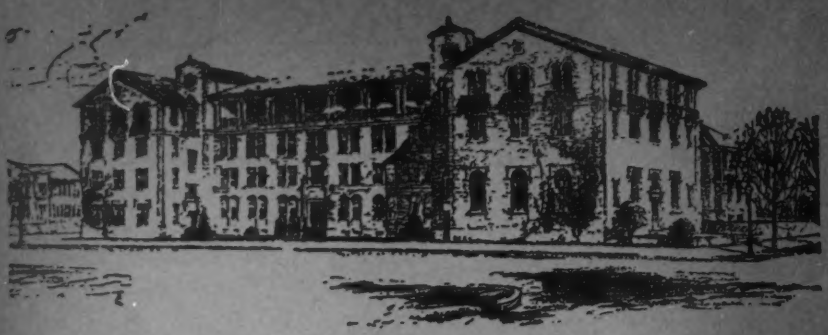
BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. Alberto P. Haedo.
Vice-Presidente: Dr. V. R. Carri.
Secretario: Dr. Renoto Segre.
Pro-Secretario: Dr. Carlos A. Gutierrez.
Tesorero: Dr. J. M. Tato.
Pro-Tesorero: Dr. Norberto Von Soubiron.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Carlos A. Cleves C.
Vice-Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Elpidio Posada.
Tresoreo: Dr. Mario Arenas A.



Central Institute for the Deaf

NATIONAL RESIDENTIAL AND DAY SCHOOL FOR THE DEAF AND DEFECTIVES IN SPEECH

Approved by Advisory Council of Foremost Ear Specialists and Educators

New fire-proof buildings beautifully located opposite Forest Park. Modern Dormitories and Equipment. Best home environments. Pupils constantly in care of teachers or experienced supervisors.

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Two years of Training following a professional curriculum for applicants with adequate college qualifications. Graduates qualify for degrees of Bachelor of Science in Education or Master of Science in Education from Washington University. Graduates prepared to teach both the deaf and speech defective.

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818 S. KINGSHIGHWAY 10, ST. LOUIS, MO.

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